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TRANSACTIONS OF THE

SECTION ON

# Practice of Medicine

of the
American Medical Association
at the Seventieth Annual
Session, held at Atlantic City,
N. J., June 9 to 13, 1919

*AMERICAN MEDICAL ASSOCIATION PRESS*
*CHICAGO: NINETEEN HUNDRED AND NINETEEN*

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# LIST OF OFFICERS

Those named below have served as officers in this Section. This Section has included other subjects in the earlier years, as will be seen by the headings which denote the different Section titles. The names of the officers have been taken from the published records, which are deficient in some cases. It will be appreciated if any additional data are brought to the attention of the Secretary of the American Medical Association.

### SECTION ON PRACTICE OF MEDICINE AND OBSTETRICS

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(Sessions Discontinued on Account of Civil War.)

1864 Chairman, B. Fordyce Barker, New York City.
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1865 Chairman, Z. Pitcher, Kalkaska, Mich.
Secretary, Ellsworth Eliot, New York City.

1866 Chairman, L. I. Tefft, Syracuse, N. Y.
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Secretary, E. Hall, New York City.

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1878 Chairman, T. F. Rochester, Buffalo.
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1879 Chairman, J. S. Lynch, Baltimore.
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1904 Chairman, Richard C. Cabot, Boston.
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1905 Chairman, Herbert C. Moffit, San Francisco.
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1906 Chairman, Thomas D. Coleman, Augusta, Calif.
Secretary, J. L. Miller, Chicago.
Delegate, J. H. Musser, Philadelphia.

1907 Chairman, C. F. Hoover, Cleveland.
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Vice Chairman, Walter L. Bierring, Des Moines, Iowa.
Secretary, Roger S. Morris, St. Louis.
Delegate, J. A. Capps, Chicago.

1912 Chairman, W. Jarvis Barlow, Los Angeles.
Vice Chairman, Thomas MacCrae, Philadelphia.
Secretary, Roger S. Morris, St. Louis.
Delegate, J. A. Capps, Chicago.

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Vice Chairman, John B. Elliott, Jr., New Orleans.
Secretary, Roger S. Morris, Clifton Springs, N. Y.
Delegate, Ray L. Wilbur, San Francisco, Calif.

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Vice Chairman, John L. Dawson, Charleston, S. C.
Secretary, Roger S. Morris, Clifton Springs, N. Y.
Delegate, Richard C. Cabot, Boston.

1915 Chairman, Roger S. Morris, Cincinnati.
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1917 Chairman, Lawrence Litchfield, Pittsburgh.
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Secretary, James S. McLester, Birmingham, Ala.
Delegate, W. J. Stone, Toledo, Ohio.

1918 Chairman, Walter L. Bierring, Des Moines, Iowa.
Vice Chairman, Charles J. Jennings, Detroit.
Secretary, James S. McLester, Birmingham, Ala.
Delegate, George D. Head, Minneapolis.

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# PROCEEDINGS OF THE SECTION

## Wednesday, June 11—Morning

The section was called to order at 9:15 by the chairman, Dr. Walter L. Bierring, Des Moines, Iowa.

Dr. Bierring read the chairman's address, entitled "Relations of Internists to Military Medicine." No discussion.

Dr. George W. McCaskey, Fort Wayne, Ind., read a paper on "The Basal Metabolism and Alimentary Hyperglycemia Tests of Thyrotoxicosis." Discussed by Drs. John C. Hemmeter, Baltimore; Lewellys F. Barker, Baltimore; Nelson W. Janney, New York, and George W. McCaskey, Fort Wayne, Ind.

Dr. Henry S. Plummer, Rochester, Minn., read a paper on "The Classification of Goiters." Discussed by Drs. Emil Goetsch, Baltimore; Lewellys F. Barker, Baltimore; Henry A. Christian, Boston; George W. McCaskey, Fort Wayne, Ind.; Charles H. Mayo, Rochester, Minn., and Henry S. Plummer, Rochester, Minn.

Dr. James P. O'Hare, Boston, read a paper on "Compatibility of Long Life with Low Renal Function." Discussed by Drs. Herman Mosenthal, New York; Edward F. Wells, Chicago; Lewis A. Conner, New York, L. D. Bulkley, New York, and James P. O'Hare, Boston.

Dr. Edward J. Wood, Wilmington, N. C., read a paper on "The Recognition of Tropical Sprue in the United States." Discussed by Drs. Douglas Vanderhoof, Richmond, Va.; Joseph H. Pratt, Boston; Walter C. Alvarez, San Francisco; Emanuel Libman, New York; M. L. Graves, Galveston, Texas; Alfred T. Livingston, Jamestown, N. Y., and Edward J. Wood, Wilmington, N. C.

The chairman appointed to serve on the executive committee, for this meeting, Drs. George Blumer, New Haven, Conn., and Charles F. Hoover, Cleveland, in the places of Drs. Roger S. Morris, Cincinnati, and Lawrence Litchfield, Pittsburgh, who were absent.

Dr. David Riesman, Philadelphia, read a paper on "Hypertension in Women." Discussed by Drs. Joseph H. Pratt, Boston; L. F. Bishop, New York; James M. Anders, Philadelphia, and David Riesman, Philadelphia.

## Thursday, June 12—Morning

Dr. Charles F. Hoover, Cleveland, read a paper on "The Functions of the Intercostal Muscles." Discussed by Drs. Henry Sewall, Denver; Charles L. Minor, Asheville, N. C., and Charles L. Hoover, Cleveland.

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Dr. Ralph Pemberton, Philadelphia, read a paper on "Chronic Arthritis in the Army: Observations Based on a Survey of Four Hundred Cases." Discussed by Drs. E. P. Joslin, Boston; Lewis A. Conner, New York, and Ralph Pemberton, Philadelphia.

Dr. Frank B. Wynn, Indianapolis, read a paper on "The Psychic Factor as an Element in Temperature Disturbance as Shown by Some Observations in the Selective Draft." Discussed by Drs. F. M. Pottenger, Monrovia, Calif.; Emanuel Libman, New York; Henry S. Plummer, Rochester, Minn.; F. B. Turck, New York; George D. Head, Minneapolis; E. F. Wells, Chicago, and Frank B. Wynn, Indianapolis.

Dr. Bertnard Smith, Los Angeles, read a paper on "The Importance of Physical Development in Cases of Effort Syndrome." Discussed by Drs. Eveleth W. Bridgeman, Baltimore; W. S. Thayer, Baltimore, and Bertnard Smith, Los Angeles.

Dr. Willard J. Stone, Toledo, Ohio, read a paper on "Pericarditis as a Complication of Pneumonia Based on Three Hundred Necropsies." Discussed by Drs. Charles S. Williamson, Chicago; Charles F. Hoover, Cleveland; William H. Robey, Jr., Boston; Henry Sewall, Denver, and Willard J. Stone, Toledo, Ohio.

Dr. Charles C. Bass, New Orleans, read a paper on "Effective and Practical Treatment of Malaria to Disinfect Infected Persons and to Prevent Relapse." Discussed by Drs. Charles S. Williamson, Chicago, and Charles C. Bass, New Orleans.

Dr. Thomas McRae and Elmer H. Funk, Philadelphia, presented a paper on "Errors in the Diagnosis of Chronic Pulmonary Tuberculosis." Discussed by Drs. Leo Kessel, New York; John A. Lichty, Pittsburgh; F. M. Pottenger, Monrovia, Calif.; A. G. Shortle, Albuquerque, N. M., and Elmer H. Funk, Philadelphia.

### Friday, June 13—Morning

The following officers were elected: chairman, Dr. James S. McLester, Birmingham, Ala.; vice chairman, Dr. Joseph H. Pratt, Boston; secretary, Dr. G. Canby Robinson, St. Louis; delegate, Dr. Roger S. Morris, Cincinnati.

The chairman, Dr. W. L. Biering, Des Moines, Iowa, said: It has been suggested that those honorary guests who are interested in the section of medicine be recommended as honorary fellows of the American Medical Association. I cannot give you the names, but if you will move that those honorary guests who are interested in internal medicine be recommended for honorary fellowships in the American Medical Association, I believe that will cover the purpose.

Motion made, seconded and carried.

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The chairman: When we were arranging this program it was suggested by the secretary that we have a war unit program to cover the divisions of medicine in the expeditionary forces. So this program is largely his work. It seems fitting and appropriate that as this work has been largely done under the supervision of Brig.-Gen. W. S. Thayer, he should preside at this symposium on the war units, and at this time we take particular pride in referring again to the distinction that has come to this section in having a member as chief consultant of the divisions of medicine in the expeditionary forces and I take pleasure in introducing to you the chairman of the morning's session, Dr. W. S. Thayer of Baltimore.

Dr. W. S. Thayer: *Mr. Chairman, Ladies and Gentlemen:* It seems to me really almost out of place at a meeting in which the program has been so remarkably well arranged and in which the general management has been so good as this, for anybody else to preside. I have been to a good many meetings of the American Medical Association, and I cannot remember one in which the program was better or in which the program has been managed so well as this.

I thought I would start in with the program this morning, indeed it was Dr. Bierring's suggestion, that I might say a few words about what this division of professional service was on the other side. It will only take a minute or two, and I have made a program or chart here. As you all know, at the time of the outbreak of our entrance into the war, our medical army corps, a small, compact body, suddenly found itself confronted with enormous problems. Just how large the problems were in France, one can hardly fancy unless he saw them there. The regular army nucleus almost to a man had to take administrative positions. All the care of the soldier, physical and medical, was handed over to members of the reserve corps. Early in the latter part of the fall of 1917 the chief surgeon established a plan of what might be called professional supervision by physicians and surgeons of the reserve corps in the form of a group of advisers or consultants, as they were finally called, who by their help were to advise in connection with the practice of medicine and surgery. Indeed, Dr. Finney and I, who were made chief consultants in surgery and medicine, were told that we should be held responsible for the character of the practice of surgery and medicine in the A. E. F. This is a pretty large responsibility for which we hardly had the means to live up to; but we did our best. There were other means: first, the surgical division presided over by Dr. Finney; second, the department of laboratories, the sanitary division, presided over by Colonel Sailer, and, third, the medical division, of which I was the head. This chart shows roughly what we attempted to do. Headquarters were at Neuve

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Château. From time to time our advice was asked in regard to the character of the medical officer, with regard to questions of their assignment to this, that or the other place. Largely our work lay in the supervision and standardization of the medical work. There were in the office first, the division consultant, Colonel Boggs, and later Major McLane. That officer was my right hand man, I think I might better have called myself his right hand man, and who was responsible for all the activities of the division, and Dr. McLane especially gave most efficient attention to the forming of medical teams, getting together men and orderlies from each base hospital who might in times of emergency be sent to the front to take care of cases of surgical shock. Each member of these teams was sent to Dijon where he took a case in surgical shock given by Colonel Cannon and his assistants. Colonel Gilman was the senior consultant in neuropsychiatry. He had been there before I was and he was the head of an important division and his work was most valuable. There were psychiatrists with each division and phychiatric consultants. There was a neurologic hospital established in the advance area near Neuve Château, where cases of psychiatry were stopped and prevented from passing to the base and later on during the Argonne offensive there was an exceedingly valuable neurologic hospital established right up in the army area amid the evacuation hospitals where these cases of fear, concussion, anxiety neuroses were stopped before they could get to the rear, rested for several days, and a large proportion sent back to their organizations. There were also consultants and psychiatrists in each hospital as in each base section. There was the senior consultant in influenza, Colonel Longcope, who did valuable work in connection with Colonel Sailer, visiting the various base hospitals and working very hard in the army area during the epidemic of influenza. The senior consultant of medicine in gas was Colonel Dexter of Cleveland. Colonel Dexter did a good deal of work in instructing the various divisions as they came in, for at the outset almost none of us knew much about poisoning by intoxicating gases. He also directed during the Argonne offensive a group of hospitals right up behind the front. He worked in laiason with the medical gas officers of the various corps and divisions which were gradually being formed at the time of the armistice. The division gas officers were not under our service but Colonel Dexter worked with them. Then there was Colonel Webb of Colorado Springs, senior consultant in tuberculosis, who when he came over visited the base hospitals and helped to standardize the work in tuberculosis and made arrangements for sending the cases of tuberculosis to the various hospitals. The senior consultant in cardiovascular diseases was Col. Alfred E. Cohn of New York, who worked not only in cases of effort syndrome but devoted himself especially to giving help and assistance to the

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establishment of convalescent camps which were in their incipiency at the time of the armistice. Through Colonel Cohn's office we sent to Dr. Lewis in England many men for instruction also a group of men epecially qualified in cardio-vascular work. We sent to the camp what cases of effort syndrome were likely to be collected. Colonel Cohn gave valuable instruction in a series of lectures in the army medical school, and those lectures were at the request of Colonel Ashworth typewritten and widely distributed. Then we had this scheme gradually improved while we were there for medical consultants to assist the chief surgeon with the object to standardize the work. There was a senior consultant and a corps consultant; one of the corps consultants was Col. George Draper of New York, who was the consultant in the fifth army corps, who will speak to you today. Then there was a plan for assistant corps consultants which was never carried out. In each base hospital as the base hospitals in France were arranged in groups, the largest groups consisting of twenty base hospitals, each one of 1,000 bed capacity with an emergency capacity of 2,000 with a convalescent camp of 7,000, so that the centers at times would contain 40,000 patients. The commander at one of the camps remarked to me that his command at one of the camps was larger at that camp than the command of the commander in chief of the U. S. Army at the time he entered the service. At each of the centers there was a consultant who helped with the commanding officer to standardize the work, and stimulated in the spirit to work, and Colonel Capps will tell you of the work that was done there. The men that came late and helped to do the work which was just begun did splendid work, and then finally we were establishing consultants in the section areas under base sections. All about through those areas were camp hospitals or small contingents were established in training areas or about large centers of different sorts, and it became necessary for some one to overlook the medical work and see that the right sort of officers were in those camp hospitals. We were just beginning to establish the medical supervision in those base areas. For instance, our secretary, Colonel McLester, had just begun his work as consultant in the base section at Brest and it was easy to see how valuable he was going to be there, when the armistice came.

That is in a rough way the picture of the work we were trying to do.

It is a great advantage to be presiding officer, and I am obliged to say one or two words before introducing the first speaker. Shortly before we went over there we were short of medical officers, and it became desirable to get officers trained in internal medicine who could take charge of the services and release senior men who could be used as consultants or could occupy positions as consultants. In looking

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around, one of the men that I most especially wanted was temporarily on service in a small Red Cross hospital. I consulted with others about the possibility of obtaining him for work and was told that this officer was unhappy where he was. He had been doing work with the British in regimental service before our entry into the war, and he felt that his work lay in the front line work. He was the type of a man who would have been extremely valuable as chief of medical service. I wanted him for that purpose, but I was told so strongly that his duty lay at the front that I did not ask for him. In a short time he returned to the front as battalion surgeon in the second division, repeatedly living almost in the trenches, going over the top with his men, living in no man's land, attending to the removal of men from the field. He went everywhere that that corps d'élite, the second division, went. On two special occasions, not to speak of many others, when we tried to get him back into the place where we felt he was better equipped in the medical standpoint, I took steps to arrange with the corps surgeon that we might obtain him for the medical position, but was met everywhere with two statements: first, that he would not if he could possibly help it be removed, and, second, that no one was willing to spare him. He was an important man. And so he went through the entire war with the second division and ended finally in the important division with regard to the question of ambulance work, and we never could remove him from the point of greatest danger. He is the kind of figure that one likes to present as the presiding officer, of the type of U. S. Army medical officer, and finally having made him uncomfortable, I am going to ask him to speak. He was recommended for every decoration and he deserved them all.

The following papers were read as a "Symposium on Medicine in the American Expeditionary Forces":

Dr. Maurice C. Pincoffs, Chicago: "The Division: Evacuation and Ambulance."

Dr. George Draper, New York: "The Corps and Army."

These two papers on "Problems at the Front" were discussed by Drs. Homer F. Swift, New York, and W. S. Thayer, Baltimore.

Dr. Joseph A. Capps, Chicago: "The Work of the Group Consultant."

Dr. Joseph Sailer, Philadelphia: "The Work of the Group Consultant."

These two papers on "Problems at the Base" were discussed by Drs. R. T. Woodyat, Chicago; Robert H. Halsey, New York, and William Darrach, New York.

Dr. Richard Dexter, Cleveland: "Immediate Recognition and Evacuation of Gas Cases."

Dr. Alfred E. Cohn, New York: "Later Effects."

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Dr. Alwin M. Pappenheimer, Hartsdale, N. Y.: "Pathology."

These three papers on "Poisonous Gases" were discussed by Drs. Maurice C. Pincoffs, Chicago; W. S. Thayer, Baltimore; M. W. Ireland, Washington, D. C., and S. J. Crumbine, Topeka, Kan.

Dr. Homer F. Swift, New York: "Trench Fever."

Dr. Warfield T. Longcope, New York: "Survey of the Epidemic of Influenza in the American Expeditionary Forces."

These two papers on "Specific Infections" were discussed by Drs. W. S. Thayer, Baltimore; Albert E. Roussel, Philadelphia; Edwin R. LeCount, Chicago; Ernest Zueblin, Cincinnati, and Homer F. Swift, New York.

Dr. Reginald Fitz, New York: "Nephritis in the Soldier." Discussed by Drs. Louis Ferl, Jr., Cincinnati; E. P. Joslin, Boston; H. E. Jones, Roanoke, Va.; Homer F. Swift, New York, and Reginald Fitz, Boston.

Dr. Marion A. Blankenhorn, Orville, Ohio: "The Treatment of Chest Injuries." Discussed by Drs. Ernest Zueblin, Cincinnati, and Marion A. Blankenhorn, Orville, Ohio.

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# RELATION OF INTERNISTS TO MILITARY MEDICINE *

WALTER L. BIERRING, M.D.
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It has been a traditional custom in the medical departments of the Army and Navy to regard the medical officers as surgeons, the director of each department being referred to as the Surgeon-General. There is but one instance in the history of the medical corps of the Army in which reference is made to a different title, the occasion being the appointment of Dr. William Brown as physician general of the middle department. Dr. Brown served from 1775 to 1780.

In the field of preventive medicine, bacteriology and sanitation, the federal medical services have a brilliant record of achievement, and many epochal contributions to scientific medicine have been made by officers of the several medical corps.

During the Civil War, Surgeon-General Hammond, well known as a physiologist and neurologist, and founder of the Army Medical Museum, created special hospitals for cardiac, pulmonary and neural diseases, at one of which Da Costa first described the condition of irritable heart in soldiers. In the Spanish-American War no distinction was made of a medical division in either base or field hospitals.

It was not until the entrance of this country into the world war that internists became more intimately related to military medicine, and it is one of my purposes in this paper to record briefly the part taken by medical reserve officers, recognized as internists, and particularly by members of this section.

* Chairman's address.

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### INTERNAL MEDICINE IN THE WAR

At the time when war was declared on Germany in April, 1917, there were twenty-nine Red Cross base hospitals ready for service, having been organized from the Medical Reserve Corps under the direction of Col. Jefferson R. Kean, M. C., U. S. Army. This number was rapidly increased until within a very short time fifty Army Red Cross hospitals were assigned for duty overseas. These base hospitals were largely representative of medical college faculty or hospital staff organizations in civil life. A medical service was an organic part of each, being in charge of a chief and usually two assistants, all well known internists. Later about ninety-four evacuation, field and mobile hospitals were organized, composed of a proportionately smaller personnel, each of which included a medical service as a part of the respective hospital.

Early in the organization of the office of the Surgeon-General of the Army, the importance of tuberculosis and cardiovascular disorders was recognized, particularly in the operation of the selective service regulations, as well as special examination of officers and men at the various military training camps.

A division of internal medicine was created under which the subdivisions for tuberculosis and cardiovascular and gastro-intestinal diseases were included. The efficient organization of this new division was due in a large measure to the experience and foresight of the first chief, Col. Theodore C. Janeway; and his untimely death in the line of duty is one of the tragic incidents of this war. The high standards set by Colonel Janeway were ably maintained by his successor, Col. W. T. Longcope, until his assignment to foreign service, when he was succeeded by Col. Lewis A. Conner, who served until the close of the war.

As the large base hospitals were established in connection with the National Army and National Guard cantonments throughout the country in 1917, the selec-

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tion of chiefs and assistant personnel for the respective medical services involved a responsibility of the highest degree, and it must have been a distinct comfort to all concerned with the health and welfare of the new army to recognize the character and standing of the internists assigned to the different stations. In a number of instances the chiefs of medical services later became the commanding officers of the Army hospitals to which they had been originally assigned.

It was the evident policy of the department to retain the regular medical corps officers in charge only long enough to permit the medical reserve officers the opportunity to become familiar with the military organization, after which the responsibility was promptly transferred to them.

### VALUABLE EXPERIENCES

The readiness with which the medical reserve officer adapted himself to the new environment was remarkable. The internist in civil practice was considered fortunate if he had supervision of a service of from fifty to a hundred beds. To be suddenly called on to assume charge of a medical service in a military hospital comprising from 1,400 to 1,800 beds, and during the influenza epidemic as high a number as 4,700 beds, required the exposition of rare tact, judgment and organizing ability. The fact that it was so successfully accomplished reflects great credit on the Medical Reserve Corps.

The opportunities offered for observing large numbers of cases of a particular disease were unique. Attention need only be directed to some of the papers presented at the 1918 session and those offered for this meeting. Reports on from 600 to 800 cases of pneumonia, 350 cases of pericarditis, 280 instances of acute meningitis, 400 cases of arthritis, and 360 cases of epidemic parotitis, occurring in a single medical service indicate the unusual responsibilities entailed and represent a condition not likely ever to occur again.

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It will no doubt be appreciated that the environment of the sick soldier as compared with the patient in civil life offered several new factors to be considered; nevertheless the accumulated records of careful observers will constitute a valuable addition to our knowledge of the particular diseases concerned.

It may be premature to indicate the gain to medical science through the experiences of military service; but even at this time it will be admitted that we have approached a little nearer to the control of respiratory infections; that attention has been more prominently directed to the extrameningeal manifestations of the meningococcus, and that a better understanding has been obtained of nephritis in the young, functional cardiac disorders, the effort syndrome and its importance as a problem in civil life, the result of inhaling poisonous gases, and the transmission and control of trench fever. The effect produced on diabetes among peoples affected principally by sugar restriction is one of the interesting revelations of the war period.

After the military hospital service was thoroughly organized, the medical officer lived under ideal conditions for the practice of medicine. Free from the embarrassment of professional competition and the financial cares of civil practice, he lived in congenial association with medical officers in charge of the surgical, roentgen ray and pathologic services, permitting daily conferences and a coordination of work contributing to the best in the diagnosis and the care of the sick.

As the medical officer, and particularly the internist, returns to civil life, it will be natural and very probable that he will wish to continue similar clinical associations at home, and thus gradually lead to a more general formation of groups of consulting specialists, a plan in keeping with the trend of modern medicine that promises much for professional development and the welfare of the public.

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The purpose of group consultants in the American Expeditionary Forces will be considered at this session, and the advantages of this feature of the service more prominently presented.

The true function of the internist is exerted in the direction of higher attainments in diagnosis, and the degree to which military medicine was advanced in that respect may be a proper measure of the wisdom of establishing a special division of internal medicine.

## MEDICAL TEACHING

In round numbers, about 150 military hospitals of the different types were established during this country's participation in the war. The fact that the department had great difficulty in finding a sufficient number of medical officers for the services in internal medicine is a commentary on the incompleteness of preparation among medical men for this special work.

In a study of the graduates of a leading medical school affecting the classes of 1915, 1916 and 1917 as to the preference expressed in the applications for commissions in the Reserve Corps, it was noted that a relatively small percentage chose the service in internal medicine; but on further investigation it was found that these usually represented the best students in the respective classes.

We are not specially concerned here with medical education as related to military conditions, recently so ably discussed by Munson and others,[1] except so far as the experiences in the services of internal medicine furnished the reason for this discussion.

The demonstrable use of examination methods, the proper interpretation of physical signs, and the ability to form diagnostic conclusions constituted the criteria of efficiency in this branch of the military service. It was in many instances the real index of the previous training of the medical officer.

1. Munson, E. L.: The Needs of Medical Education as Revealed by the War, J. A. M. A. **72**: 1050 (April 12) 1919. Vaughan, V. C.: Correspondence Department, ibid., p. 1095. Foster, N. B.: Medical Education as Revealed by the War, ibid. **72**: 1540 (May 24) 1919.

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Nevertheless it reflects great credit on the medical resources of this country that it was possible in one brief year to develop the School of Military Medicine at Fort Oglethorpe, where in October, 1918, 3,000 medical officers and 30,000 medical corps men were being trained.

That it was possible by short courses of intensive instruction to remedy some of the defects in medical training recognized in the general practitioner on admission to military service was probably due to the fine cooperative spirit and ideal conditions for teaching that prevailed in the military medical school. That it could so readily be done was indicative, at least, of a good fundamental education.

If the experience of medical teaching in the war has directed attention to an improvement in the scheme of education for the better practice of medicine, it will have offset in part the great sacrifices it has otherwise entailed.

### INTERNISTS IN THE NAVY

It is fitting at this time to make reference to the internists afloat. At the beginning of the war the Medical Department of the Navy recognized the great need of internists, particularly in the large base hospitals in the naval training stations. Reserve officers were placed in charge of the different services of internal medicine, having supervision also of the clinical laboratory; and as rapidly as possible a similar plan was carried out on all ship hospitals and base hospitals overseas.

As the internists in the Naval Reserve are gradually being returned to civil life, Surgeon-General Braisted has endeavored to meet the occasion by requiring of medical officers of the Navy attendance at the Navy Medical School for a three months' course each in the branches of surgery, laboratory and clinical diagnosis, and the head specialties to determine fitness for special service, indicating that the new departure inaugurated during the war is to be continued.

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The entire transport service was in charge of the Navy, and the completeness with which old vessels were transformed into hospital ships with every equipment for the care of sick soldiers and sailors testified to the initiative and organizing ability of the Navy Medical Corps. When the story is fully told of the devotion and sacrifice of medical officers in connection with influenza epidemics aboard ships, it will constitute one of the heroic chapters of the war.

### DISTINGUISHED SERVICES

It is not possible at this time to recall the many examples of heroism displayed by the medical reserve officers; but there is a source of pride in directing attention to a few instances, and furthermore to the manner in which the medical officer measured up to the demands of entirely new duties.

Reference has been made to the assignment of internists as commanding officers of cantonment base hospitals in this country, and evacuating hospitals in the American Expeditionary Forces, indicating that they had recognized administrative ability. The medical officers, among whom were several well-known internists who received the Croix de Guerre, form a large list.

It is quite impossible to estimate in any appreciative sense the service to the country of Colonel Billings in establishing the division for the rehabilitation of the disabled soldier and sailor.

In investing Brigadier-General Thayer with the Distinguished Service Medal, the citation read:

> As chief consultant in medicine of the A. E. F. it was largely through his individual efforts that the treatment of the sick was so standardized, coordinated, and proficiently perfected as to result in a direct saving of many lives and a consequent conservation of man power and morale of these forces.

One of the contributors to the war unit program of this section applied for active service, April 6, 1917, and went to France with the Second Division. In civil

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life he is associated with one of the prominent medical clinics of the country. With practically no surgical training, his entire military service was concerned with the charge of stretcher bearers and dressing stations at the front.

In regard to Capt. M. C. Pincoffs, the Division Surgeon, Lieut.-Col. Richard Derby reports that:

> His services have been of a high order of excellence. He possesses great initiative, and has performed every duty required of him with vigor and thoroughness. He has maintained excellent discipline and morale among his subordinates. He has time and again demonstrated his utter fearlessness and disregard of personal safety in the various engagements in which the Second Division has participated, as evidenced by his having been recommended for the Distinguished Service Cross, the Medal of Honor, and the Croix de Guerre.

As an example of versatility of talent I would like to refer to a member of the section who in civil life is chief of a department of medicine in a large university medical school, and was inducted into active service a few days after contributing a paper on pericarditis at the 1917 session in New York. During a military service of twenty-one months he probably did not make a single heart or lung examination because his activities were directed in an entirely different direction. He became the chief of the school for sanitary instruction at the Fort Riley medical officers' training camp, and when the military medical training was later concentrated at Fort Oglethorpe, he was assigned to a similar service there. He developed an open air school of military hygiene including every form of apparatus and equipment for demonstration, which permitted every student officer to gain a comprehensive knowledge of the subject in a short period of time. One of his distinct contributions was an incinerator capable of disposing of 10 tons of garbage with a minimum of expense, which has been adopted by the Medical Department of the Army. By reason of his training as a clinician, Colonel Williamson was able to direct the instruction in sanitary medicine, of particular value to

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the military service and of incalculable benefit to the thousands of medical officers that came under his charge and their future usefulness in civil practice.

When the medical history of this war is written, the record of the internist will indicate an important part in the efficient accomplishments of the several medical services during the great conflict.

In conclusion I beg to express my appreciation for the courtesy extended by the section in selecting me as chairman for this session, and I feel particularly obligated to Dr. J. S. McLester, the secretary, to Dr. J. H. Pratt, the acting secretary, and all who have so kindly cooperated in the program, to which we now invite your interest and attention.

Equitable Building.

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# THE BASAL METABOLISM AND HYPERGLYCEMIC TESTS OF HYPERTHYROIDISM

## WITH SPECIAL REFERENCE TO MILD AND LATENT CASES

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A recent writer[1] has remarked that our position in the study of thyroid disease is about what it would be in regard to diabetes if we were without our laboratory data relative to the urine and the blood. It is well known[2] that in some very severe cases of thyrotoxicosis the thyroid gland is not definitely palpable, or at most not more so than in very many presumably healthy persons. This emphasizes the fundamental importance, if any emphasis were needed, of looking beyond the goiter for clinical data more distinctive and more strongly diagnostic than the clinical signs and symptoms usually present in thyroid intoxication.

Recent observations have confirmed the suspicion long entertained that other endocrine glands besides the thyroid play a more or less important rôle in exophthalmic goiter. This is especially true with reference to the thymus, the importance of which would almost seem to justify the term "thyroid-thymus syndrome" for this condition. Whether or not this term is too cumbersome, or possibly might too much exaggerate the relative importance of the thymus, it reflects a point of view which needs empha-

1. Du Bois, E. F.: Metabolism in Exophthalmic Goiter, Arch. Int. Med. 17: 915 (June) 1916.
2. McGarrison, Robert: The Thyroid Gland in Health and Disease, New York, William Wood & Co., 1917.

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sis at the present time. For instance, Blackford and Freligh,[3] on the basis of seventy-four necropsies, conclude that an enlarged thymus is present in 100 per cent. of patients under 40 years of age, and in about 50 per cent. of those above 40, in whom the average weight of the thymus was 13 gm. Matti[4] says that the monopoly enjoyed by the thyroid in exophthalmic goiter is at an end. Halsted[5] on the basis of a very large experience concludes that the important rôle played by the thymus in exophthalmic goiter is demonstrated beyond question by animal experimentation, and especially by the cures resulting from primary thymectomy and also from secondary thymectomy following thyroidectomy which alone had been unsuccessful.

There are two pressing needs in the study of thyroid disease. The first of these is the differential diagnosis of the so-called borderline cases and the determination of the toxicity of an existing definite goiter. Practically all cases of thyroid disease belong to this group at the outset for a variable period, which may be very short or very long—may in fact be measured by days or by years. A group of more or less vague symptoms, which might be either constant or intermittent, and consisting for the most part of tachycardia, nervousness, and hyperidrosis, and a trifle later varying grades of debility and digestive disorders, and perhaps a little later still, mild, irregular febrile disturbances, formed the basis of the clinical diagnosis. These symptoms are, obviously, the expression of a toxemia with somewhat variable group reactions, and which may in many cases easily be due to any one of several different causes, and especially chronic infections.

3. Blackford, J. M., and Freligh, W. P.: The Thymus in Adults with Especial Reference to Goiter, Collected Papers of Mayo Clinic **8**: 507, 1916.

4. Matti, H.: Die Beziehungen der Thymus zum Morbus Basedowii, Berl. klin. Wchnschr. **51**: 1365 (July 20) 1914, abstr. by J. A. M. A. **63**: 812 (Aug. 29) 1914.

5. Halsted, W. S.: The Thymus in Exophthalmic Goiter, Bull. Johns Hopkins Hosp. **25**: 223, 1914.

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The second pressing need is something besides the subjective sensations of the patient and the ordinary clinical manifestations, to enable us more accurately to measure the actual rise and fall of the thyrotoxic state, just as by the rise and fall of hyperglycemia and glycosuria in diabetes we follow the course of that disease. Owing to the neuropsychopathic character of so many of these thyrotoxic patients, it is essential that it should be objective in its nature. In my opinion the recent clinical application of the basal metabolism and alimentary hyperglycemia tests has very satisfactorily met these demands and marks an epoch in the study of hyperthyroidism.

## BASAL METABOLISM

We will first consider the basal metabolism which is by far the more important of these two methods. By basal metabolism is meant that minimal quantity of metabolic change essential to the neuromuscular and secretory phenomena of what might be called the basal and necessarily continuous organic functions—respiration, circulation and secretion. Rest and food abstinence approximately eliminate all other metabolic activities. It has been well known since the classical work of Friedrich Müller[6] and Magnus-Levy,[7] about twenty-five years ago, that one of the most characteristic phenomena of hyperthyroidism was an increase in general metabolism, while hypothyroidism produced exactly the opposite result. This, of course, depends on the regulatory action of the thyroid secretion on metabolic processes, which apparently bear a direct relationship to each other. It is perfectly evident, therefore, that the optimum conditions of body function are dependent on a definite quantity of thyroid and synergistic secretions which can only vary within

6. Müller, Friedrich: Beiträge zur Kenntniss der Basedowischen Krankheit, Deutsch. Arch. f. klin. Med. **51**: 335, 1893.

7. Magnus-Levy: Gaswechsel bei Thyroidea, Berl. klin. Wchnschr., **32**: 650, 1895.

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narrow limits without producing more or less serious disturbance.

Since that time a great deal of work has been done by various investigators in confirmation and elaboration of these facts. This work has for the most part been done in well-equipped hospitals and institutions with large and complicated apparatus requiring, for instance, the services of an expert gas analyst and, therefore, not at all practical for general clinical purposes. Taking advantage of the fact that heat production could be measured equally well if not more accurately by oxygen consumption alone than by direct calorimetry, Dr. F. G. Benedict[8] has devised a portable and comparatively inexpensive apparatus which can be operated in any small hospital or office by any one reasonably familiar with laboratory technic. The introduction of this apparatus, which can be placed in the corner of any convenient room, the only accessory being a couch, places the entire medical profession under additional obligation to this distinguished investigator.[9]

The estimation of metabolism by measuring the units of heat production is made possible by the application of Rubner's law, that it is proportional to the body surface from which, with some exceptions, all heat not actually expended in energy production is radiated. The basis of this law is, of course, the

8. Benedict, F. G.: A Portable Respiration Apparatus for Clinical Use, Boston M. and S. J. **178**: 667 (May 16) 1918.

9. Dr. Janney of New York in his discussion of this paper raised a question which I overlooked in the closing discussion, and which I think deserves attention. He said that the metabolism observation even with the Benedict portable respiration apparatus was not easy. Certainly it is not easy. The greatest care and accuracy are required. For example, if the patient after resting half an hour should, without the knowledge of the clinician, arise from the couch and walk, say 20 feet, and close a window, it would raise basal metabolism about 15 per cent. If the metabolism observation was made after instead of before the 100 gm. of glucose, in the technic described in this paper, this would cause an increase of 5 per cent. The slightest error in measuring exactly the duration of the observation, or escape of air or oxygen around the mouth piece, would introduce substantial error. A failure to correct volume of oxygen to zero C. and sea-level barometric pressure, if such carelessness were conceivable, would make another increase of from 10 to 15 per cent. It will thus be readily seen that if careless or incompetent work should find its way into literature, under a mistaken impression of the simplicity of this procedure, it would be very deplorable.

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universally accepted laws of the conservation and transmutation of energy and the radiation of heat. The chemical forces of the body, which represent the sum total of metabolism, are transmuted into heat, the excess of which over body requirements is radiated from the surface. The standard chosen is the number of calories per square meter of body surface per hour. The body surface is determined very simply by what is known as the "height-weight formula" based on two factors: namely, the height in centimeters and the weight in kilograms. By the use of the chart, plotted from this formula, as worked out by DuBois,[10] and the two factors of weight and height, the body surface expressed in square meters is instantly read without any calculation whatever. In the determination of oxygen consumption the patient is made to fast overnight and then to lie on a couch for thirty minutes or more to get rid of the respirable products of all metabolism other than basal, resulting especially from the voluntary musculature. The patient breathes through the mouth-piece connected with the open air for a few minutes in order to establish quiet normal breathing, after which the breathing is in and out of a spirometer bell. The oxygen and nitrogen only are rebreathed, for the carbonic acid is entirely removed from the expired air by being passed through an absorbent (soda-lime). The scale on the side of the spirometer bell is read at the beginning of the observation and again at the end of whatever period is chosen (usually ten minutes), both observations being made at the same point of time; that is, at the end of expiration. Since the carbonic acid gas has disappeared and the nitrogen is not influenced, the difference in the readings on the spirometer scale accurately represent the amount of oxygen consumed. The volume of gas is then corrected for barometric pressure and

10. Du Bois, Delafield, and Du Bois, E. F.: A Formula to Estimate the Approximate Surface Area if Height and Weight Be Known, Arch. Int. Med. 17: 863 (June) 1916.

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thermometer readings in order to bring it to the standard of sea-level and zero centigrade. This corrected volume which is obtained by the use of logarithms, but which could undoubtedly be determined on the percentage basis with sufficient accuracy for all clinical purposes, is used as the basis of the indirect calorimetric calculation. Each liter of oxygen gas, with the above mentioned corrections, represents 4.825 calories of heat. From this the calories are calculated for one hour. By dividing the calories per hour by the body surface, determined as above indicated, we have the final result of calories per square meter per hour.

The number of calories per square meter of body surface per hour varies normally with sex and age and perhaps other physiologic conditions. By the Du Bois or "height-weight" formula, the average figure for men is 39.7; for women, 36.9. In pathologic conditions and conspicuously in disturbances of thyroid secretion, these figures are subject to numerous variations. In hyperthyroidism the increase is striking; very mild cases, including the borderline group, and frank cases of thyrotoxicosis in the quiescent state, may show an increase up to 30 per cent.; mild cases, 30 to 50 per cent.; severe cases, 50 to 75 per cent., and very severe cases, 75 to 100 per cent. or more, following the classification of Du Bois excepting that the "mild" cases of Du Bois (below 50 per cent. increase of basal metabolisms) are subdivided into "mild" and "very mild" to correspond with his "severe" and "very severe" cases above 50 per cent. increase.

Table 1 and Chart 1 illustrate the range of cases studied and give the general grouping of the cases. Perhaps the most important feature is the relatively large proportion of cases belonging to the mild and very mild or borderline group. As indicated above, the main purpose of this clinical study was the differential diagnosis and positive identification of the cases in this group, which frequently cannot be done by the

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ordinary clinical methods. There will be found, with some exceptions, a striking parallelism between the percentage increase of basal metabolism and the relative intensity of the thyrotoxicosis. This parallelism is so close that the percentage increase of the basal metabolism seems to serve as a reliable guide in the classification of the cases and an accurate measure of the fluctuations that may occur either spontaneously or as a result of treatment.

TABLE 1.—PARALLEL OBSERVATIONS ON BASAL METABOLISM AND BLOOD SUGAR IN ACTUAL AND SUSPECTED CASES OF HYPERTHYROIDISM

| Case | | Age | Calories per Sq. Meter per Hour | Percentage of Variation from Normal | Blood Sugar | | |
|---|---|---|---|---|---|---|---|
| | | | | | Fasting | First Hour | Second Hour |
| 1. Mrs. M. | 1263 | 60 | 35.2 | — 4.6 | 0.125 | 0.140 | 0.119 |
| 2. Mr. S. | 1775 | 44 | 37.9 | — 4.5 | 0.089 | 0.105 | 0.098 |
| 3. Mrs. F. | 1765 | 68 | 36.5 | 0 | | | |
| 4. Mrs. T. | 1455½ | 38 | 37.2 | 0 | 0.770 | 0.090 | 0.136 |
| 5. Mr. S. | 1747 | 38 | 40.7 | + 2.5 | 0.087 | 0.226 | 0.258 |
| 6. Miss J. | 1776 | 24 | 39.0 | + 5.7 | 0.078 | 0.120 | 0.110 |
| 7. Miss K. | 1727 | 27 | 39.9 | + 8.1 | 0.112 | 0.129 | 0.113 |
| 8. Mrs. R. | 907 | .. | 40.0 | + 8.4 | 0.089 | 0.139 | 0.113 |
| 9. Mr. R. | 1778 | 33 | 43.7 | +10.0 | | | |
| 10. Mr. S. | 8 | 54 | 45.0 | +13.3 | 0.090 | 0.150 | 0.121 |
| 11. Mrs. K. | 1378 | 25 | 42.0 | +13.8 | 0.071 | 0.072 | 0.092 |
| 12. Mrs. E. | 1582 | 50 | 42.1 | +14.0 | | | |
| 13. Mrs. E. | 1740 | 34 | 43.7 | +18.4 | 0.110 | 0.146 | 0.138 |
| 14. Mrs. Y. | 1761 | 41 | 45.1 | +22.2 | 0.084 | 0.194 | 0.072 |
| 15. Miss W. | 1723 | 35 | 45.9 | +24.4 | 0.094 | 0.233 | 0.161 |
| 16. Mrs. Y. | 1700 | 43 | 48.3 | +30.9 | 0.120 | 0.140 | 0.132 |
| 17. Mr. B. | 1726 | 28 | 56.3 | +41.9 | | | |
| 18. Mrs. C. | 1708 | 32 | 55.9 | +51.5 | 0.065 | 0.210 | 0.167 |
| 19. Mrs. H. | 1752 | 35 | 59.0 | +60.0 | 0.092 | 0.195 | 0.178 |
| 20. Mrs. B. | 1549 | 29 | 64.8 | +75.6 | 0.090 | 0.220 | |

In the study of basal metabolism it is important to remember that like all physiologic processes in either normal or pathologic conditions, it is subject to marked variations. This variation, however, offers no greater difficulties here than in other procedures, such as the tests for albuminuria, glycosuria, the Wassermann test, variations in temperature, etc. When properly correlated with the general clinical syndrome it may be regarded as one of the most reliable and scientifically accurate aids available in any field of diagnosis, and is of inestimable value not only in establishing the diagnosis but in studying the clinical course of this disease.

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### ALIMENTARY HYPERGLYCEMIA

The occurrence of alimentary hyperglycemia in hyperthyroidism has been known since the work of Tachau[11] in 1911. At that time and until quite recently, the methods of blood analysis were too cumbersome for clinical use. The recent investigations of Lewis and Benedict,[12] Myers and Bailey,[13] and others, however, placed this work on an entirely different and

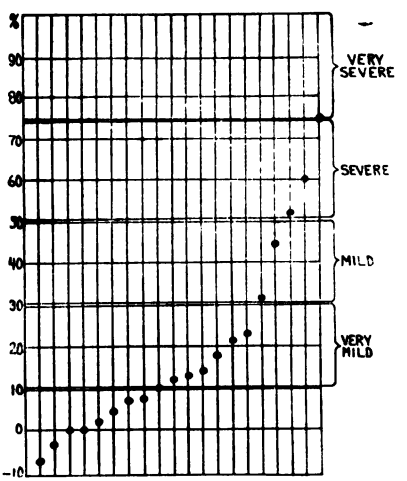


Chart 1.—Basal metabolism in actual and suspected hyperthyroidism.

clinically practical basis, and stimulated both experimental and clinical investigations along this line. With the aid of these very simple and accurate colorimetric methods the entire subject of blood sugar content has been worked over afresh within the last few years.

Living in a thyroid zone and confronted almost daily by the diagnostic difficulties above mentioned, I was attracted by the method described in the paper by Denis, Aub, and Minot.[14] This method seemed entirely

11. Tachau: Deutsch. Archiv. f. klin. Med. **104**: 445, 1911.
12. Lewis and Benedict: J. Biol. Chem. **20**: 61, 1915.
13. Myers and Bailey: J. Biol. Chem. **24**: 147, 1916.
14. Denis, W.; Aub, J. C., and Minot, A. S.: Blood Sugar in Hyperthyroidism, Arch. Int. Med. **20**: 964 (Dec.) 1917.

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practical. The test, as outlined by them, can be completed in a little more than two hours, or if immediately preceded by the basal metabolism observation, which is my method of choice, the entire procedure will require less than three hours, most of this time being spent in waiting.

The fasting blood is first taken; then the patient is given 100 gm. of glucose. In one hour a second blood specimen is taken, and in another hour a third, which by this technic completes the blood study. The urine

TABLE 2.—EVIDENCE OF NO HYPERGLYCEMIA IN NON-HYPERTHYROID CASES

| Case | | Fasting | After 100 Gm. Glucose | | Remarks |
|---|---|---|---|---|---|
| | | | 1st Hour | 2d Hour | |
| Mr. F. | 1655 | 0.065 | 0.080 | 0.107 | |
| Mr. S. | 1775 | 0.098 | 0.105 | 0.093 | Doubtful |
| Miss A. | 1175 | 0.125 | 0.100 | 0.100 | |
| Mrs. H. | 1386 | 0.096 | 0.094 | 0.095 | |
| Miss M. | 1627 | 0.095 | 0.090 | 0.100 | |
| Mr. B. | 1660 | 0.077 | 0.080 | 0.082 | |
| Mr. S. | 1711 | 0.065 | 0.065 | 0.060 | |
| Mr. M. | 1608 | 0.073 | 0.061 | 0.067 | |
| Miss K.* | 1727 | 0.112 | 0.129 | 0.113 | |
| Mrs. A. | 1751 | 0.069 | 0.097 | 0.091 | |
| Miss R. | 1707 | 0.090 | 0.091 | 0.088 | |
| Mrs. M.* | 1263 | 0.125 | 0.140 | 0.119 | |
| Mrs. K. | 1378 | 0.071 | 0.072 | 0.092 | |

* Cases 1263 and 1727 show an absolute hyperglycemia, but it will be noted that the fasting blood sugar is quite high, raising a question of an associated pathology.

also is examined at the end of the first and second hours, and may be examined for several hours longer, although the glycosuria is a question involving the renal glucose threshold rather than the thyroid question.

Under normal conditions, in the average person in the fasting state, 100 gm. of glucose does not produce a hyperglycemia lasting as long as one hour. At least, this was the result in fourteen of my own cases, and in sixteen of those recorded by Denis, Aub, and Minot.[14] Hamman and Hirschman,[15] however, obtained

15. Hamman, L., and Hirschman, I. I.: Studies on Blood Sugar, Arch. Int. Med. **20**: 761 (Nov.) 1917.

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an alimentary hyperglycemia in normal persons following 100 gm. of glucose, the crest of the wave occurring in from twenty to forty minutes but still remaining to some extent at the end of one hour or longer. In view of these facts, it must be regarded as possible that a transient hyperglycemia occurred during the first hour in observations made according to the methods used in this paper, disappearing at the end of the first hour.

Whatever may be the fact in regard to normal persons, there seems to be no question that in hyperthyroidism after the ingestion of 100 gm. of glucose there is almost invariably a marked hyperglycemia, the crest of the wave occurring at the end of the first hour, or at least recognized then by this method, with a sharp fall in 70 per cent. of my cases by the end of the second hour. This curve was found in twenty-two of thirty-one cases (about 70 per cent.) of hyperthyroidism. In six cases (20 per cent.) the hyperglycemia continued to rise until the end of the second hour. In three cases only (or about 10 per cent.) the hyperglycemia did not occur until the end of the second hour. These curves are represented graphically in Chart 2, based on Tables 3 and 4. The delayed hyperglycemia shown by the curves represented in Types 2 and 3 is probably the result of retarded absorption due to gastro-intestinal conditions. It suggests the possibility of a belated hyperglycemia occurring in some of the cases in which it does not appear within the two hour period. This is apparently a valid criticism of this technic. My answer is that in the forty-five cases of actual or suspected hyperthyroidism forming the basis of this section of this paper, there were thirteen (Table 2) which did not show a hyperglycemia within two hours, and in none of these could the diagnosis of hyperthyroidism be established on other grounds, although two or three still look suspicious.

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In practically every case in which the diagnosis of hyperthyroidism seemed to be reasonably well established on other grounds, including the basal metabolism observations, a definite hyperglycemia occurred not later than the second hour, and in 90 per cent. of the cases within the first hour. As a routine procedure, therefore, I believe that the two hour period is sufficient.

In doubtful cases, and especially when the determination of basal metabolism cannot be made, the

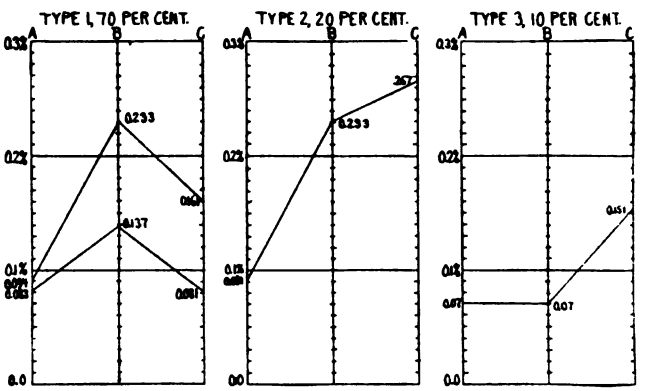


Chart 2.—Sample curves illustrating types of alimentary hyperglycemia in thirty-one cases of hyperthyroidism: *A*, blood sugar fasting; *B*, blood sugar one hour after 100 gm. of glucose; *C*, blood sugar two hours after 100 gm. of glucose.

alimentary hyperglycemia test, as above outlined, may prove to be of considerable value. Of course diabetes, alcoholism, etc.,[16] must be excluded.

## SUMMARY

The differential diagnosis of many mild, atypical or very early cases of thyrotoxicosis by means of the ordinary clinical signs and symptoms can only be made with varying degrees of probability. The need is,

16. In view of Pemberton's very valuable contribution made at this session, we must add arthritis to the list, and further studies may possibly add still more. The alimentary hyperglycemia test can only be regarded, then, as confirmatory, our main reliance being placed on basal metabolism.

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therefore, very great for definite objective data, either decisive or very strongly corroborative, not only to determine diagnosis, but to estimate therapeutic results.

TABLE 3.—EVIDENCE OF HYPERGLYCEMIA IN LATENT BUT SUSPECTED HYPERTHYROIDISM

| Case | | Fasting | After 100 Gm. Glucose | | Remarks |
|---|---|---|---|---|---|
| | | | 1st Hour | 2d Hour | |
| Miss H. | 766 | 0.066 | 0.190 | 0.118 | |
| Miss S. | 1602 | 0.075 | 0.190 | 0.210 | |
| Miss H. | 1654 | 0.070 | 0.140 | 0.128 | |
| Mrs. F. | 1616 | 0.070 | 0.140 | 0.140 | |
| Mr. A. | 1570 | 0.900 | 0.168 | 0.146 | |
| Mrs. H. | 1640 | 0.060 | 0.200 | 0.100 | |
| Mr. W. | 1641 | ..... | 0.116 | 0.174 | |
| Mrs. T. | 1644 | 0.066 | 0.122 | 0.134 | |
| Mr. B. | 1664 | 0.091 | 0.233 | 0.267 | Maximum increase |
| Mr. B. | 1663 | 0.070 | 0.070 | 0.151 | |
| Miss B. | 1673 | 0.075 | 0.061 | 0.131 | |
| Mrs. P. | 1721 | 0.132 | 0.191 | 0.138 | |
| Mrs. F. | 1612 | 0.060 | 0.149 | 0.144 | |
| Mr. B. | 1724 | 0.096 | 0.200 | 0.175 | |
| Mrs. G. | 1699 | 0.099 | 0.138 | 0.068 | |
| Mrs. S. | 1733 | 0.063 | 0.137 | 0.061 | |
| Mrs. Y. | 1761 | 0.064 | 0.194 | 0.072 | |
| Mrs. R. | 907 | 0.069 | 0.139 | 0.113 | |
| Mrs. T. | 1455½ | 0.077 | 0.090 | 0.136 | |
| Mr. S. | 1747 | 0.087 | 0.226 | 0.258 | |
| Mrs. F. | 1740 | 0.110 | 0.146 | 0.138 | Minimum increase, 33% |

TABLE 4.—EVIDENCE OF HYPERGLYCEMIA IN FRANK HYPERTHYROIDISM

| Case | | Fasting | After 100 Gm. Glucose | | Remarks |
|---|---|---|---|---|---|
| | | | 1st Hour | 2d Hour | |
| Mr. C. | 1649 | 0.060 | 0.128 | 0.080 | |
| Mr. S. | 1684 | 0.065 | 0.086 | 0.065 | Postoperative |
| Mr. S. | 8 | 0.090 | 0.150 | 0.121 | |
| | | 0.078 | 0.176 | 0.113 | |
| | | 0.090 | 0.220 | | |
| Mrs. B. | 1549 | 0.054 | 0.180 | 0.076 | Highest increase 233% |
| Mrs. G. | 1597 | 0.090 | 0.126 | 0.129 | Postoperative |
| | | | 0.063 | 0.100 | At end of rest treatment period |
| Miss C. | 1708 | 0.065 | 0.210 | 0.167 | |
| Mrs. L. | 1719 | 0.063 | 0.157 | 0.135 | |
| Miss W. | 1723 | 0.148 | 0.208 | | |
| | | 0.094 | 0.233 | 0.161 | |
| Miss H. | 1752 | 0.092 | 0.195 | 0.178 | |
| Miss J. | 1776 | 0.078 | 0.120 | 0.110 | |
| Mr. C. | 1649 | 0.089 | 0.165 | 0.130 | |
| | | 0.070 | 0.096 | 0.075 | Postoperative |
| Mrs. G. | 1597 | 0.054 | 0.180 | 0.076 | |

The clinical application of basal metabolism and alimentary hyperglycemia satisfies these demands in a very scientific, practical and satisfactory manner.

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Basal metabolism, or the metabolism of the fasting body in the resting state, can be quickly and accurately determined by measuring the oxygen consumption with the Benedict portable respiration apparatus. The average normal heat production, which is an accurate index of metabolism, is about 39.7 calories for men and 36.9 for women by the "height-weight" formula per square meter per hour under the conditions indicated. Physiologic variations of not more than 10 per cent., and nearly always much less than this, may occur in either direction.

In hyperthyroidism there is an increase up to 100 per cent. or more, according to the severity of the intoxication, which varies in different cases, and in the same case at different times.

In hyperthyroidism there is a diminished tolerance of carbohydrates with alimentary hyperglycemia, and also with glycosuria whenever the hyperglycemia exceeds the renal glucose threshold of the individual case.

In every case of hyperthyroidism studied (thirty-one cases) the blood sugar content was increased within two hours from 50 to 200 per cent.

In 70 per cent. of thirty-one cases the maximal rise occurred at the end of the first hour, with a more or less sharp decline at the end of the second hour, proving that the crest had been reached and passed.

The very few cases in which hyperglycemia is highest at the second hour may be explained by gastric hypomotility and slow intestinal absorption.

The failure of the hyperglycemia to rise proportionately to the increased metabolism and to bear any direct relationship to the intensity of the thyrotoxicosis suggests that it is an indirect phenomenon due perhaps to overexcitation of other organs, for example, the pancreas, which in exceptional cases fails to respond to the thyroid stimulation.

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The application of these tests will probably reveal a much greater incidence of hyperthyroidism than has formerly been recognized, and will especially enable us to make a clear diagnosis between toxic and nontoxic goiters. While a final diagnosis is altogether a question of clinical judgment, and should not be made exclusively in the laboratory; and while neither increased metabolism nor alimentary hyperglycemia is pathognomonic of hyperthyroidism, yet, when rationally correlated with the remaining clinical data, they together make as clear-cut a diagnosis of hyperthyroidism as a positive Wassermann does of syphilis in a suspected case.

I wish to acknowledge my indebtedness to Drs. J. B. Rieger, B. M. Edlavitch, and K. M. Whitten for their assistance in this work.

## ABSTRACT OF DISCUSSION

Dr. John C. Hemmeter, Baltimore: It is a great pleasure to listen to a paper that aims to have physiologic facts for its foundations. Dr. McCaskey's aim is simply to afford a new and practical method of diagnosis in doubtful cases of diseases of the thyroid. He assumes that the processes of oxidation in hyperthyroidism are increased and that there is a hyperglycemia two hours after 100 gm. of glucose are injected into the circulation and that this hyperglycemia is pathognomonic. I have seen the apparatus Dr. McCaskey employed for the respiring of a measured amount of oxygen by a resting subject. I saw no means for the absorption of the humidity that the patient himself gives off to the expired air. It makes a good deal of difference in the carbon dioxid absorbed by lime and the patient's own humidity. If it increases that of the respired gas to 80 it will increase the percentage of blood sugar by 13 per cent. of the normal. I would suggest some provision to account for this change in the blood sugar due to the humidity gradually accumulating in the spirometer, i. e., if the expired air actually reenters the spirometer. I do not wish to go through all the arguments of sugar metabolism from Claude Bernard, Pavy and Otto Cohnheim and our own men down to Folin, Frederick M. Allen and others, but I hold that when you inject 100 gm. of sugar into an individual, a hyperglycemia two hours thereafter is physiologic. Therefore the method requires revision and further investigation. H. J. Hamburger (Groningen, Holland) in the most recent investigations which have come

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to me in the last ten days is opposed to this view of colloidal blood sugar. He says that all sugar when injected into the animals that have a separate circulation in the glomeruli from that of the renal tubules, as in the frog, behaves like sodium chlorid, and he gives further evidence that sugar exists in the blood just like sodium chlorid—not in combination. Postprandial hyperglycemia can be found in 90 per cent. of normal people. That there is an excess of blood sugar that can be caused by injecting 100 gm. of dextrose directly into the circulation can be ascertained by using several of the most modern methods for blood sugar determination. The whole matter has been gone over by McCleod in his recent book on biologic chemistry as applied to medicine. The excessive oxidation in hyperthyroidism appears to be well established by many biochemists. But in this very fact when associated with hyperglycemia we have a paradox and that paradox is one of the most puzzling things in medicine. Here we have a furnace that will burn anthracite coal and refuses to burn paper although you blow in pure oxygen. Of all the foods the carbohydrate molecule is the easiest to split up under above conditions, and the organization splits up the protein molecule, the most difficult, and even oxidizes the carbohydrate molecule moiety of the proteid molecule. It reminds one of a simile from a verse in the "Ancient Mariner"—"Water, water, every where, nor any drop to drink"—"Sugar, sugar every where, and not a milligram to oxidize."

Dr. Lewellys F. Barker, Baltimore: These two tests—that for measuring basal metabolism and that for measuring the glucose content of the blood—are certainly valuable tests in clinical diagnosis, especially for deciding upon the existence of what we call "overactivity of the thyroid gland." The methods, to the uninitiated, sound complex. They are, in reality, fairly easy to perform; and it is interesting that clinical men have devised simple modes of making measurements that formerly involved a very complicated technic. The basal metabolism is certainly increased in severe forms of exophthalmic goiter, and the sugar content of the blood, also, seems to be increased. Dr. Hemmeter has suggested that the existence of a hyperglycemia implies a state in which the body cannot split the carbohydrate molecule. May it not rather be simply that the sugar molecule, in some states at least, is mobilized more quickly than under normal conditions. For if the body, in the state under consideration, can split, as he says, the protein molecule and can burn the carbohydrate moiety of this protein molecule, why should it not be able, in the same state, to split the sugar molecule itself. The hyperglycemia might then be looked upon as the result, merely, of a sudden and rapid mobilization of sugar. Whether the increased basal metabolism and the hyperglycemia are due to the direct action

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of thyroid products, or are indirect results of thyroid activity, remains to be seen. We must think of the influence of the thyroid products on the cell metabolism all over the body, on the one hand; and, on the other, we must keep in mind the influence of the thyroid products on the activity of the other endocrine glands. I would mention one relationship, especially that of the thyroid to the suprarenal gland. Many of the phenomena that we speak of as hyperthyroidism may, in reality, be due to hypersuprarenalism, and it may be that some of our clinical tests for thyroid activity measure directly rather the suprarenal activity, and only indirectly the thyroid influence. I simply point out some of the difficulties, though I think that the tests are really valuable in internal medicine and I hope that they will be much more employed, and that we may come to some agreement as to what the results they yield may mean.

Dr. Nelson W. Janney, New York: With regard to the blood sugar tolerance test it has been the experience of myself and co-workers that by standardizing the technic it is possible to get more definite and accurate results than previously obtained. We now give, as already reported, 1.50 gm. sugar dissolved in 2.5 c.c. of water per kilogram of the patient's body weight, adding the juice of one lemon for flavoring. The fasting blood sugar value is obtained immediately before and one and two hours after taking the sugar lemonade. We thus find the normal blood sugar level to run from 0.12 to 0.14 per cent. without glycosuria. I wish to call attention to a very important point not yet presented in this discussion, namely a criticism of the value of the blood glucose tolerance test in diagnosis. Its value in this respect is problematical for myself and others have repeatedly observed abnormally high blood sugar curves in various conditions, i. e., pituitary disorders, Addison's disease, and other less clearly defined endocrinopathies. Unfortunately, therefore, this test, though frequently indicating an abnormal carbohydrate metabolism in hyperthyroidism, is by no means pathognomonic of that condition. We should, I feel, await with caution and patience more extended observations in this most interesting field.

Dr. George W. McCaskey, Fort Wayne, Ind.: In regard to Dr. Hemmeter's remarks, no matter whose figures he quotes, 0.3 per cent. as the normal blood sugar has certainly been discredited entirely. This figure was obtained by imperfect methods in use at that time. It has only been possible in the last few years by the use of the colorimeter to clinically determine the blood sugar content accurately. I believe we can do it now very closely. As to the factor of humidity in metabolism observations, I am not a physicist, but have made all the corrections indicated by Dr. Benedict. The fact that in forty-five consecutive cases the fasting blood sugar con-

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tent is about what it ought to be, shows that there was no gross error, and even admitting any error, this error will be the same in both fasting and alimentary glycemic test, and that is all that is really necessary for the purposes of this clinical study, as it makes the comparative values reliable. Dr. Barker, I think, hit the nail on the head when he said that the hyperglycemia is not directly caused by the thyroid excess. I believe that the thyroid with other synergistic members of the endocrine system may modify other functions, such as the internal secretion of the pancreas, or the glycogenic or storage function of the liver, and in that way account for changes in blood sugar content. It is not simply a question of oxidation. It may be, among other things, mobilization, as Dr. Barker said. A failure on the part of the storage function of the liver or the voluntary musculature may account for the hyperglycemia. Dr. Jamey's criticism that a certain amount of glucose should be given per kilogram of body weight is well taken and is probably important. As a matter of fact, however, it did not seem to modify clinical results. Dr. Geyelin found glycosuria when the glucose intake exceeded 1.8 gm. per kilogram of body weight per hour. This is not a thyroid question at all, but a question of the renal glucose threshold, which Denis, Aub and Minot have established at about 0.17 or 0.18 per cent. blood sugar. Undoubtedly the scientific way to do this is to give a certain amount of sugar per kilogram of body weight, although in none of my thirteen normal cases did a hyperglycemia occur with a uniform intake of 100 gm. of glucose irrespective of body weight, unless the fasting blood sugar was already high. This is probably best accounted for by the enormous reserve function which apparently enables a patient weighing, say one hundred pounds, to promptly metabolize 100 gm. of glucose as readily as a patient weighing 150 pounds. It is perfectly obvious that if the glucose were increased, a point would be reached at which one patient could metabolize it and the other could not.

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# COMPATIBILITY OF LONG LIFE WITH LOW RENAL FUNCTION *

JAMES P. O'HARE, M.D.
BOSTON

It is customary to consider chronic nephritis as a steadily progressive, degenerative process. As a rule, we do not think of the possibility of a stay in its progress when the disease has reached an advanced stage. One would certainly predict in such a case that the duration of life, with renal function near zero, must be extremely short. And yet, at the Peter Bent Brigham Hospital we have observed several patients who have lived for three or four years with such a low renal function. I wish here to report two such cases.

In the time allotted, no complete study or detailed description of the cases is possible. In order to tell as much as possible I have tried to make the chart self-explanatory.

## REPORT OF CASES

CASE 1.—A girl who, at 9, had scarlet fever without known sequelae; at 18 and 19 had a severe anemia necessitating her being in bed for three months and one month, respectively. In view of subsequent events I think we can fairly assume that our patient had an unrecognized acute nephritis following her scarlet fever, and at 18 and 19, acute exacerbations of a chronic nephritis. So far as we could learn, there was no other possible etiology.

About this time she began to develop vascular symptoms which took the following forms: spasmodic blurring of sight; cramps in legs and fingers, and occasional dizzy spells. Morning headaches then began to appear with increasing frequency, severity and duration. In addition there were occasional drowsy spells. Apart from the latter, our patient's symptoms had been entirely vascular and not at all renal. At the age of 23 (in May, 1915) she entered the hospital for her headaches.

* From the medical clinic of the Peter Bent Brigham Hospital.

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Physical examination at that time revealed a moderate secondary anemia; a normal heart; no evident sclerosis of peripheral or retinal vessels; a blood pressure of 165—110; no edema, and negative eye grounds.

The urine was of low gravity, and contained a slight trace of albumin, but no casts. (It is important to note that at no time during our three and one-quarter years of observation did the urine show any sign of an active degeneration in the kidney. Hyaline casts even were very rare.) The phenolsulphonephthalein excretion was only 12 per cent., instead of the normal 60 to 80 per cent. The blood urea nitrogen was 59 mg. per hundred c.c., about four times the normal. The renal test diet showed a marked inability on the part of the kidneys to handle water, salt and nitrogen.

During the patient's six weeks' stay in the hospital, her general condition improved, the headaches were relieved, and the blood pressure fell to normal. Her renal condition, as far as we could measure it, did not change.

It would seem fair to conclude that the vascular condition with intermittent hypertension was responsible for the chief symptom—headache—and that at this time the kidneys, though marked sclerosed, were given her comparatively little trouble in spite of their low function. I do not believe, however, that any of us felt at the time that the patient would live for three and one-quarter years with such a low renal function.

In the sixteen months previous to her second entry in November, 1916, the patient was fairly well. Her headaches, however, returned; nosebleeds were common, and there was an occasional drowsy spell. Her symptoms were still largely vascular. She came back to the hospital chiefly for study.

This showed the following changes: (1) definite cardiac hypertrophy; (2) a return to her hypertension; (3) some sclerosis of the retinal arteries (though none could be detected in the radials and brachials), and (4) a few white spots (probably degenerative) in the retina. The urine and all of the functional tests were identical with those at the previous entry. There was no evidence of active renal degeneration, and renal function was near the minimum.

It would seem fair to conclude that the vascular disease was progressing with an effect on the heart and retina but no measurable change in the kidneys.

During 1917 the patient was not seen, but was apparently very well except for her headaches. She went about, took long walks, danced, etc. In December she had the grip, apparently without much effect. In February, 1918, she had an attack suggesting renal colic with hematuria. About the first of April she caught a severe cold, which was followed by a very severe headache, vomiting, much blurring of vision and edema of face, neck and upper sternum.

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At entrance this time she showed the following new findings: (1) the tender edema mentioned above; (2) a heart of the same size but with more signs of hypertrophy; (3) definitely palpable radials; (4) greater sclerosis of retinal arteries with small hemorrhages in each eye; (5) a higher blood pressure, 190 and 120; (6) the phenolsulphonephthalein excretion had dropped to 0, where it stayed until her death, eight months later, and her blood urea nitrogen had climbed to 98 mg.

In the hospital she began to improve rapidly. Her blood pressure began to fall. She then developed an acute infection

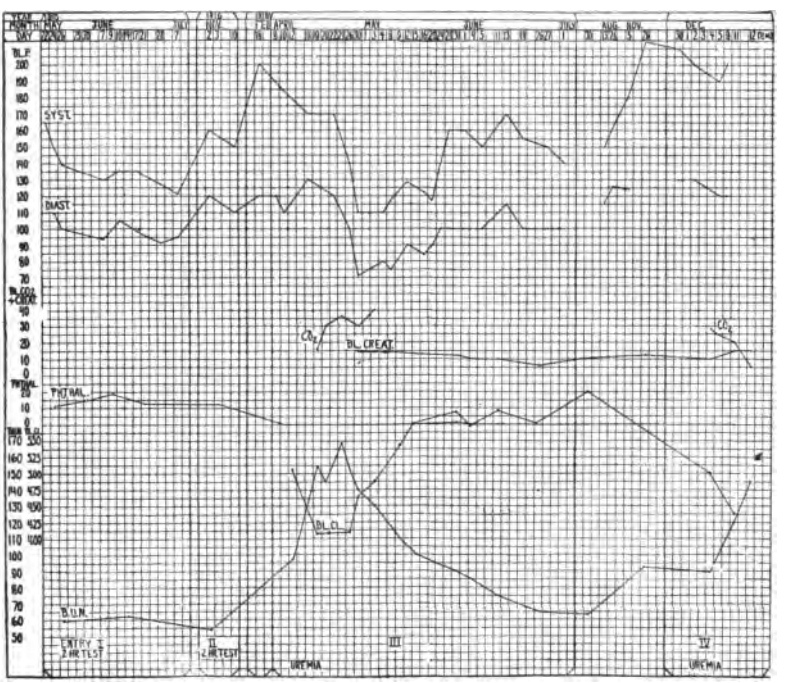


Chart 1.—Case 1: *Syst.*, systolic blood pressure; *Diast.*, diastolic blood pressure; $CO_2$, bicarbonate combining power of the plasma; *Bl. Creat.*, creatinin of the blood; *Phthal.*, phenolsulphonephthalein excretion; *N. P. N.*, nonprotein nitrogen of the blood; *Cl.*, or *Bl. Cl.*, blood chlorid; *B. U. N.*, blood urea nitrogen.

of the antrum, and with this went immediately into uremia with drowsiness, twitching, two severe convulsions and coma. Her blood pressure did not rise and soon after showed a fall to a very low normal. There was a marked acidosis, the blood carbon dioxid reaching a level of 16 mm. The blood urea nitrogen rose rapidly to 168 and the blood chlorid fell from 5.05 gm. liter to 4.20. This drop may have been due to an

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infusion of sodium bicarbonate, although we know that the blood chlorid is frequently low in uremia. With the improvement in the acidosis, the blood chlorid rose to a high level. The patient then began to improve, although she was restless and twitched for three weeks. The acidosis quickly disappeared and the blood urea nitrogen fell very gradually to near its former level—70 mg. It is of interest to note that the creatinin of the blood at this time was high, 14 mg. This is supposed to indicate death within a short time, and yet the patient lived eight months. Before her discharge in July her blood pressure gradually rose, with the improvement in her general condition, to a level slightly above normal.

We can reasonably infer that at this entry her renal function, which had been sufficient up to now, had begun to give way as a result of an acute infection. She entered in impending uremia but started to improve until a new infection plunged her into an outspoken severe uremia.

In the five months previous to her last entry, Nov. 30, 1918, the patient was fairly well. Her old symptoms persisted and her blood pressure climbed rapidly to well over 200. Then to her vascular renal symptoms she began to add cardiac ones—dyspnea and angina pectoris. There were occasional periods of low grade uremia, too, and her strength was poor. On reporting to our renal class in November, her blood urea nitrogen was found to be over 90 mg., and she was consequently sent into the hospital.

The findings at this time were similar to those at her last entry except that the radials were very tortuous; the retina showed fresh exudate and hemorrhages; blood pressure was much higher and there was twitching of the arms and legs. All of the laboratory findings were the same except that the blood urea nitrogen was higher. The blood chlorid had fallen considerably from the last estimation in July.

In the hospital she steadily went down hill. Twitching became more prominent, but there were no convulsions. Nausea and vomiting became extreme. Edema developed around her jaws. We noted a pleurisy (which was found to be superimposed on a pneumonia), and noted also what seemed to be a pericardial rub. This proved to be endocardial and due to fresh vegetations on the aortic and mitral valves. In the meantime the blood pressure had dropped some. Acidosis, as indicated by the blood carbon dioxid, developed. The blood chlorid fell (as a result either of her pneumonia or her uremia), and her blood urea nitrogen rose to 120 mg. before death.

At necropsy the kidneys were the smallest we had ever seen. The two together weighed about 60 to 65 gm. They showed a well marked vascular nephritis. The renal arteries were not abnormal except that the aortic orifices were almost closed by atheroma; but the vessels within the kidney were quite prominent. The cortex was only 2 mm. in thickness.

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Under the microscope there was practically no kidney substance left. The arteries, large and small, were all affected, showing thickening, narrowing and even obliteration of many of the smaller ones. The glomeruli were extremely rare and showed all sorts of changes from slight hyalinization of the capillary wall to complete thrombosis of the whole tuft. Some were just compact masses covered with epithelium. The subtended tubules were mostly atrophied. There was much connective tissue. The sclerosed areas alternated with those where the glomeruli, though few, were fairly normal and connected with hypertrophied, apparently functioning, tubules.

Here, then, is presented a case of nephritis which, starting presumably from scarlet fever at 9, had gone on for eighteen years, passing through various phases. Nine and ten years after the assumed onset the patient had what we believe to have been acute exacerbations of a chronic nephritis. Coincidently with, or subsequent to, these attacks the vascular apparatus of the body—including the kidney—began to play an ever-increasing part. When the patient first came under our observation, fourteen years after the onset of the disease, she had a markedly sclerosed kidney with a very poor renal function. Just how poor this was can be readily seen. Three years before her death the two hour renal test showed very advanced disease in the kidney. Her excretion of phenolsulphonephthalein in these years has only once been as high as 18 per cent., and for the last year of her existence has been practically 0. Her blood urea nitrogen has always been over 50 mg., about four times the average value. During the last year her blood creatinin has always been around 10 mg. And yet, in spite of this extreme renal impairment, she got along very well with these kidneys for three and one-quarter years. This was undoubtedly due to the fact that the process in the renal parenchyma had become inactive and that nothing happened to light it up. The only possible changes within the kidney were the slow intravascular ones which could not be measured. By living carefully within the low limits set by the few functionating

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glomeruli and tubules, our patient lasted much longer than could have been expected.

The important thing to bear in mind is that for the last ten years of the patient's life, the disease had been almost entirely a vascular one with greater vascular change outside the kidney than within. The renal function, while low, was sufficient until infections broke the narrow thread by which it maintained itself.

CASE 2.—An old man on whom we have data for eleven years before his death in 1919 (in fact, we have records of normal urinalyses at the Boston City Hospital as far back as 1891 and 1893) on account of his age, arteriosclerosis, and other complicating factors, presented symptoms that were more complex and the development of which it was more difficult to trace. The apparent onset of his nephritis was in January, 1908, when he was 62 years old. He had a "bad cold" followed by swelling of his eyes and face. He was in bed intermittently for eight weeks, part of the time in the Boston City Hospital. Here the records showed albuminuria and hematuria. About the first of June the edema had increased, and there were dyspnea and orthopnea. This necessitated his entry into the Massachusetts General Hospital, where he stayed for two weeks. The diagnosis of subacute glomerulonephritis, ascites, double hydrothorax and cirrhosis was made. The urine showed a large trace of albumin, a few hyaline and granular casts, and a moderate number of red and white cells. The blood pressure, determined for the first time, was found to be 150.

The patient was not seen again for three years, but was apparently well until just before his next visit to the Massachusetts General Hospital in October, 1911. At this time the diagnosis was chronic nephritis and cardiac decompensation. His urine still showed signs of some renal activity. His blood pressure, as can be seen on the chart, had increased to 190—125. The first phenolsulphonephthalein test showed an excretion of 17 per cent. in one hour.

The patient was seen a few times in the outpatient department of the Massachusetts General Hospital in 1911 and 1913. In 1914 he had a flareup of his nephritis following an acute diarrhea.

Shortly afterward, in April, 1914, he first entered the Brigham Hospital (Chart 2). His condition was very poor. There was an acute exacerbation of his nephritis, and uremia was impending. He was stuporous, drowsy, nauseated and had headaches. His blood pressure was over 200. His phenolsulphonephthalein excretion was 0, and his nonprotein nitrogen climbed to 130 mg. per hundred c.c., about five times

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its average amount. Furthermore, his added urea and salt test showed marked inability to handle these two substances.

The outlook with such findings was without doubt grave. Certainly one could not expect him to live five more years, as he did.

He was seen a few times in 1914 in the outdoor department for headache, weakness, dyspnea and drowsiness. In March, 1915, he entered our hospital a second time for these symptoms. He seemed to be in a state of low grade uremia. His urine was about the same, except that there was no activity.

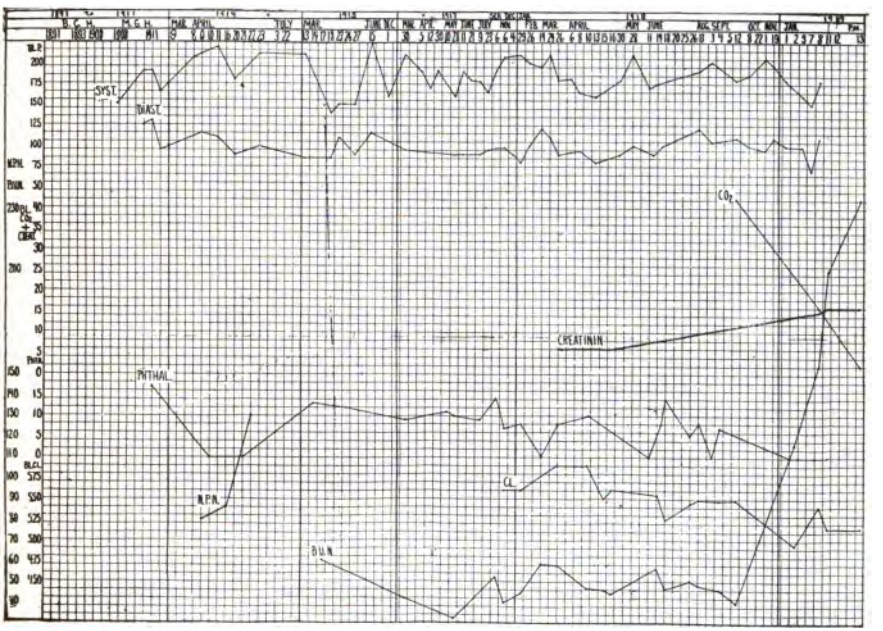


Chart 2.—Case 2; abbreviations same as in Chart 1.

His blood pressure was still over 200, but rather promptly fell to normal, as can be seen on the chart. The phenolsulphonephthalein had improved a bit, but was still extremely low (13 per cent.). The blood urea nitrogen was over 60 mg. Without doubt, at this time the patient's kidneys were markedly sclerosed.

He appeared at the outdoor department occasionally during 1916, 1917 and early 1918. During this time his urine showed but slight activity. His pressure varied from 225 to 160 systolic, with the diastolic more steady around 100. The phenolsulphonephthalein excretion was only once as high as

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14 per cent., and the blood urea nitrogen was between 40 and 60 mg. In the early part of 1918, a two-hour renal test showed a marked fixation of all elements.

In April, 1918, he entered the Brigham Hospital the third time for slight toxic symptoms. He was not very sick and his stay was short. His urine showed no change. His blood pressure had dropped to nearly normal, his phenolsulphonephthalein and blood urea nitrogen were about the same as before. He returned in June for two weeks because of a "convulsion" and great drowsiness. There was little new, however, to be found. His urine was no different. His blood pressure was a little higher, around 175.

In September of that year he was admitted for the fifth time for severe dyspnea, palpitation and occasional twitching. The dyspnea was due to a chest full of fluid and was relieved by tapping. His blood pressure was again over 200, his phenolsulphonephthalein practically 0, and the blood urea nitrogen between 40 and 50 mg.

Between the fifth and last entries there was nothing especially new except that his heart began to give more trouble, with occasional sudden dyspnea and tightness in his chest. In November, definite alternation of the pulse was noted.

Dec. 21, 1918, he entered for the sixth time, because of precordial distress. He did not seem awfully sick but quickly became more and more drowsy. His respirations grew gradually slower and deeper. He became irrational, semiconscious and finally comatose. Twitching of hands, feet and mouth occurred, but no convulsions. There was anuria for three days. In the meantime the blood pressure dropped, as it frequently does before death. A marked acidosis developed, the blood urea nitrogen mounted quickly to 200 mg. and the phenolsulphonephthalein was, of course, 0.

At necropsy the kidneys were very small (62 and 70 gm.) red and granular. The cortex measured only 2 to 3 mm. The renal artery and larger branches were not abnormal. Microscopically, however, the vessels within the kidney showed marked arteriosclerotic changes. Many of them showed all stages of obliteration. There was very little kidney parenchyma left. The intact glomeruli were extremely scarce. Many showed recent infarction from thrombosis of the vessels leading to them. Tubules had disappeared in great numbers. Those that were left showed degeneration of all kinds. The connective tissue was particularly prominent.

### COMMENT

Here, then, is a case of chronic nephritis which, starting apparently with an acute process in 1908, had active flareups during 1911 and 1914. By the latter year the process was a very severe one with the func-

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tion of the kidney practically nil. After that the activity of the degenerative process was comparatively slight.

I want especially to call attention to the fact that during these five years the phenolsulphonephthalein excretion was never over 14 per cent., and most of the time much nearer 0, and that the blood urea nitrogen was consistently four or five times the average normal.

It is difficult to determine just why this patient survived for five years with such an extremely low function. In contrast to the carefully regulated life of the first patient, who lived within the low level set by her renal function, this patient did everything that a renal patient should not do—at least up to 1917. He was just a tramp, who ate whatever he could get and, before 1918, drank heavily. He was, however, surprisingly free from infections which might break down what little functioning tissue remained. One must conclude that the small amount of renal tissue which the patient had during the last five years was sufficient to keep him alive until the vascular disease within the kidneys had progressed to such an extent that in the last few weeks many of the small vessels became thrombosed, and with this the glomeruli supplied were put out of commission.

These two cases, then, demonstrate the possibility of prolonged existence with an exceedingly small amount of renal tissue and an extremely low renal function. The only possible explanation is that the disease during this time was a purely vascular one. The pneumonia played directly or indirectly an important part in the death of the first patient. The thrombotic obliteration of essential glomeruli was responsible for that of the second.

It can hardly be said that the true prognosis was in any way indicated by the tests performed.

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## ABSTRACT OF DISCUSSION

DR. HERMAN O. MOSENTHAL, New York: The cases Dr. O'Hare described are of great interest and importance. He made mention of the fact that most of us would not have expected these patients to live as long as they did and I believe we can readily agree with that statement. The problem before us is to find out what factors determine the prognosis in nephritis and to apply this knowledge in the treatment of our patients. The expectancy of life of the sufferer with chronic nephritis may be regarded as being measured by three possible terminations: First, factors which may be considered to be entirely extrarenal, of which heart failure, apoplexy and intercurrent infections constitute those most constantly met. It is our inability to gage their progress satisfactorily that is largely responsible for the uncertain prognosis in chronic nephritis. Second, many patients with chronic nephritis die of uremia before renal function has diminished to such a degree as to render renal insufficiency any more than a contributing factor. Often the phthalein excretion lies between 20 and 30 per cent. and the blood urea nitrogen at a level of from 40 to 50 mg. per hundred cubic centimeters of blood. It is probable that this state of affairs is a toxemia, but from what tissue this poison originates, what its nature is, and what the exact conditions are that call it into action are all open questions. The sudden rise of blood nitrogen seen in one of Dr. O'Hare's cases may possibly indicate an excessive protein destruction which I believe is characteristic of certain phases of the toxemia under discussion. Third, if all the above vicissitudes leave the subject of chronic nephritis alive he finally develops an increasing renal insufficiency to which he succumbs. This is a very prolonged process and it is almost incredible how low renal function may drop and the patient maintain at least fair health. Dr. O'Hare's report illustrates this very well. I have seen some similar cases though more of them were followed for as long a period. In another condition, polycystic kidney, in which an uncomplicated renal insufficiency develops, the same phenomenon of prolonged life with a maximal impairment of renal function is often found. As far as prognosis is concerned, it becomes evident that only very few nephritic patients really die as the result of renal insufficiency but that most of them cease to live because of extrarenal influences.

DR. EDWARD F. WELLS, Chicago: Satisfactory management of the patient depends largely on taking him into full confidence as to the diagnosis, the prognosis, and the general plan of treatment. Chronic interstitial nephritis is not a disease of the kidneys alone, but the blood vessels, heart, lymphatics, and other tissues are so essentially implicated that it is impossible to separate them. It takes a profound

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clinician to estimate within wide limits, the excretory capacity, under stress, of the interstitial nephritic kidney; and it requires a wiser physician than most of us to state how long any one of these patients may live. I have seen patients in whom the output of urea was extremely small for many successive months, in whom, when placed on some extra strain the kidneys have passed such quantities of urea as are beyond ordinary credence. Therefore, our prognosis should be extremely circumspect. Our first examination of a chronic interstitial nephritic patient should be made with painstaking care, and accuracy. The renal capacity should be estimated as closely as possible from several points of view, as, e. g., the excretion of water, of salt, the rapidity and completeness of the passage through the circulatory system, including the lymphatic circulation, of titratable solutions, and of the output of some of the waste products of metabolism. I value highly the measurement of the capacity of the kidneys to excrete urea, and always include this as an important feature of the investigation. The dietetic, regimental and medicinal measures which we employ may, probably, assist the kidney in its functions. However, some adventitious conditions are beyond our control as, e. g., the entrance into the system of an acute infection. In my own experience these most frequently transform, by disturbing the circulatory balance, a satisfactory progress to a condition from which the patient rarely completely recovers, and is often the beginning of a more or less prolonged downward course, ending fatally. Under these circumstances the patient will become dyspneic, and apneic; dependent edema will attract attention. It is absolutely necessary for the patient to maintain a posture as nearly horizontal as possible for a prolonged period, at least six weeks.

Dr. Lewis A. Conner, New York: The second of Dr. O'Hare's cases illustrates a point which is of great practical importance. That is, that in aged people the usual standards of normal kidney function are not applicable. If we attempt to prognosticate, in the case of people of 70 or 80 years of age, in terms of the standards of renal function which we apply to younger people, we are likely to make embarrassing mistakes. In such patients very low readings of the phthalein test and very high figures for blood nitrogen, creatinin, etc., are compatable with fairly good health and with many years of life.

Dr. L. Duncan Bulkley, New York: I have a patient who has had albuminuria for forty-nine years. In 1870 I had an acute attack of albuminuria; I had a second one soon afterward. After several years I went to the Yellowstone Park and there had a third, severe attack. Since that time my urine has been examined frequently, and albumen and various casts invariably have been found.

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DR. JAMES P. O'HARE, Boston: This type of case is a rare condition. We do not see it very often but we do see it often enough to warrant hesitancy in making a prognosis in a chronically sclerosed kidney. We have had also a few cases of the edematous chronic glomerular nephritis and the patients have lived for quite a considerable period, that is, for months, with a very low renal function, and then have died from a pneumonia. I was glad to hear Dr. Mosenthal comment on the fact that death in the nephritic is very frequently due to causes not essentially nephritic but to other conditions like cardiac failure and infection. I have often been very much disappointed in cases that we had studied for a long time to find the patient dying of an acute infection or a cardiac complication when we were looking for a pure nephritis at necropsy. It is important for all of us to make records of our observations on these cases so that if a patient does live for a longer period than we expected, we can analyze the data obtained. Most of us do not do that. In fact, there has been no opportunity to do that until now because the renal tests were unknown until a few years ago. But now we have tests which are easy to make, and it is important to use them and make observations of these patients. Bear in mind, however, that even with our functional tests we are unable at present to make accurate prognoses. We make them with a fair degree of accuracy, but that degree of accuracy is often brought about by the fact that the patients die of an intercurrent infection or of cardiac failure rather than from the kidney disease.

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# THE RECOGNITION OF TROPICAL SPRUE IN THE UNITED STATES

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There can no longer be any doubt of the existence of tropical sprue in the United States. While the majority of the cases are occurring in the Southern states, it is interesting to note that one of my series came from New Hampshire, one was seen in Syracuse (a Porto Rican case), and one came from the Johns Hopkins Hospital. It is evident, therefore, that a familiarity with its cardinal symptoms would be helpful to many more in the profession than those of us from the South.

In another communication[1] I attempted to collect all references in the literature to the occurrence of the disease in America. Since 1915, when that paper was written, the evidence has grown that the disease is by no means rare and that the cases are widely distributed. In North Carolina its occurrence is no longer a medical curiosity.

## SYMPTOMS OF SPRUE

Sprue is characterized by three groups of symptoms: those of the mouth, the intestinal tract, and the blood.

The mouth symptoms have been painstakingly described by Sir Patrick Manson,[2] who divides them into seven subheads. His description is too full to allow of even an abstract here. My own observation

1. Wood, E. J.: The Occurrence of Sprue in the United States, Am. J. M. Sc. **110**: 692 (Nov.) 1915; Tr. A. Am. Phys. **30**: 505, 1915.
2. Manson, Sir Patrick: Sprue or Psilosis, Allbutt's System of Medicine, Ed. 7, **3**: 774.

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leads me to avoid too fine a line of differentiation of these symptoms. Suffice it to say that the tongue is inflamed and pink, with congested fungiform papillae, eroded patches and superficial cracks on the dorsum and edges. Crombie's molar ulcer is frequently mentioned as a helpful symptom, but I have observed it even more frequently in pellagra. The tongue in sprue is quite different from the tongue in pellagra. In the latter disease it will be found more pointed and not so flabby. In sprue, it is very much paler than in pellagra. There is also, in sprue, an approach to the cobblestone appearance which does not occur in pellagra. After a short experience with the two diseases, any observer may expect to make a correct differentiation by examining the tongue alone.

The diarrhea in sprue is the most distinctive symptom. The bowel movements occur from midnight until about 10 o'clock in the morning. There then occurs a cessation until the next day. If the cessation does not occur, there will be, at least, a marked diminution in the number of movements in the afternoon and early hours of the night. The bowel movements are very large, suggesting at once a pancreatic condition. The reaction is decidedly acid, and there is much gas mixed with the feces. Examination of the feces reveals a large amount of fat. The stools are light in color and give a positive reaction for hydrobilirubin. Pratt and Spooner,[3] with the Schmidt-Strassburger test diet, found a fat loss of 45 per cent. and a nitrogen loss of 15 per cent. P. H. Bahr,[4] in his Ceylon cases, found a fat absorption of from 70 to 90 per cent., which is much greater than was shown in our experience. In addition to this failure in the utilization of fat and nitrogen, there are other evidences of pancreatic insuf-

3. Pratt, J. H., and Spooner, L. H.: A Study of Sprue, J. A. M. A. 59: 218 (July 20) 1912.
4. Bahr, P. H.: Tr. Soc. Trop. Med. and Hyg., London, 1914, p. 161.

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ficiency indicated by the thymus nucleus test and the Sahli glutoid salol capsule test.

The question of the relationship of the pancreas cannot be discussed here, but it is hoped that an interest in this fascinating problem may be aroused and that the material so close to hand may be utilized in the solution of an important problem. The difficulties of the problem are enhanced by a marked variation in the degree of pancreatic digestion, and also in the utilization of fat and nitrogen.

### BLOOD PICTURE

The most interesting phase of the study of sprue at this time is the blood picture. In a large number of cases the color index is above 1, and a case now under observation gives a persistent index of 1.66. The anemia has always been regarded as secondary, and I have no wish to question this point; but it is important to show how the condition may be confused with pernicious anemia. I believe that many cases of sprue have been called pernicious anemia. On the other hand, some cases of pernicious anemia have been diagnosed as sprue. One such experience has befallen me.

In sprue I have noted the most marked variation in the size of the red cells. There was found in a recently studied case a decided preponderance of oversized cells, as well as numerous dwarf cells. There also occurs poikilocytosis. It was noted that there were many very pale cells in the field when the blood was stained with any of the eosinates of methylene blue. Stipple cells were very rarely found. In the case referred to above, in which the blood was carefully studied on different occasions in many slides, no nucleated red cells were found.

Notes of a case now under observation will be briefly presented, chiefly on account of the blood picture.

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REPORT OF CASE

CASE.—Mrs. H., aged 37, the mother of three children, was married to a saw mill worker moving from point to point in North Carolina, and living under rather unfavorable hygienic conditions.

The present trouble is said to have begun at the birth of her third child in September, 1918. She denied ever having had any form of anemia before. Her physician's information was limited because of the fact of her moving about at short intervals from one saw mill to another. Following the birth of the third child she had a rather severe influenza and has never been well since.

Owing to a sore mouth and tongue, in addition to extensive pyorrhea, she was advised to have all her teeth removed, which she did.

Throughout the winter the diarrhea, which was of the matutinal character, and the anemia persisted. There seemed to have been only the very slightest variation in the severity of either symptom.

The physical examination was practically negative. The liver dulness was slightly reduced. The spleen was not palpable. The patient had lost some flesh.

The red blood count was 600,000; the hemoglobin, 20 per cent.; the color index, 1.66. No megaloblasts nor normoblasts were found on repeated examination of many slides. There were no stipple cells. There was a marked difference in the way in which the red cells took the stain. This was shown chiefly in numerous strikingly pale cells. There was a preponderance of oversized red cells, the percentage ranging from 25 to 30. The fragility test of the red cells with hypotonic salt solutions showed that hemolysis had begun at the 0.45 per cent. point and was complete at the 0.3 per cent. point. This we regarded as normal.

The white blood count was 3,200; polymorphonuclears, 40 per cent.; large lymphocytes, 16 per cent.; small lymphocytes, 44 per cent. No parasites of malaria were found. The blood Wassermann test was negative.

There was an absence of free hydrochloric acid in the stomach contents, and marked stasis.

The feces were light yellow, and were very acid in reaction. Crystals and droplets of fat were found on microscopic examination, after staining with sudan III. The beef nucleus test showed that while the nuclei of the muscle fibers were in great measure digested, there persisted nuclei which had not been completely digested. The agar tubes of Einhorn failed to give returns from which any conclusions could be drawn, probably because of faulty technic. The three-day test diet was given, and the examination to determine the absorption of fat and nitrogen undertaken; but they cannot be reported on now.

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There was no tenesmus or blood with the diarrhea. The movements were for the most part large.

### RELATION OF SPRUE TO ANEMIA

Biermer's[5] original report on pernicious anemia mentioned, as a group of symptoms, digestive disturbances with occasional diarrhea. Elder and Matthews[6] reported two fatal cases of pernicious anemia following labor. The attack was ushered in with ulceration of the tongue and throat. The work of Hunter[7] will be recalled because of his rather extreme view that streptococcic infection of the tongue and upper alimentary tract bore a part in the etiology of pernicious anemia. G. A. Charlton[8] studied the effects of chronic infection with *Bacillus coli,* and produced a very striking anemia, which was characterized by a pari passu reduction in hemoglobin and red corpuscles. He concludes that by subinfection the ordinary organisms of the intestinal tract may produce a very definite anemia.

These instances from the literature are mentioned because our study of sprue has led us to think that it may be a bridge between the anemias experimentally produced by the colon bacillus, and possibly others of the usual intestinal flora, and an anemia which probably owes its origin to a true intestinal disease, one which more nearly approaches pernicious anemia than any of the anemias produced experimentally. We are now preparing for a study of the colon bacillus isolated from the duodenal contents in sprue.

It is worthy of emphasis that the remissions in sprue are as striking as in pernicious anemia. That remarkable recurrence which is so often seen in pernicious anemia, even after years of quiescence, is also seen in sprue.

5. Biermer: Cor.-Bl. f. schweiz. Aerzte, quoted in full in Nothnagel, American Edition, 1905, Alfred Stengel, editor.
6. Elder and Matthews: Lancet **2**: 371 (Aug. 8) 1903.
7. Hunter, William: Pernicious Anemia, London, C. Griffin & Co., 1915, p. 1901.
8. Charlton, G. A.: J. M. Res. **8**: 344 (Nov.) 1902; in Nothnagel (Footnote 5).

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What the relation between the two diseases is, if any, remains to be seen, but it is not improbable that a more accurate study of sprue will shed light on many obscure corners in the study of pernicious anemia.

There is no longer an occasion to mention the confusion between sprue and pellagra. The matter should be dismissed with a word. If the sprue stool and tongue are considered, even in the light of an absence of the skin lesion, there should be no confusion.

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## ABSTRACT OF DISCUSSION

DR. DOUGLAS VANDERHOOF, Richmond, Va.: Much credit is due Dr. Wood for having called attention time and again to the existence of so-called tropical sprue in the United States. Chronic or recurring diarrhea, that is, characterized subjectively by absence of pain and tenesmus and objectively by presence of blood, mucus and pus in the stools, is generally due to one of four causes: first, achylia gastrica with diarrhea, a matutinal diarrhea. This cause of diarrhea accounts for from 90 to 95 per cent. of the cases of chronic or recurring diarrhea characterized by absence of pain and tenesmus, by blood, pus and mucus in the stools. Second, cases of pancreatic achylia, characterized by absence or deficiency of the pancreatic ferment in the duodenal contents or in the stools, perfectly definite cases, not occurring very frequently but worthy of consideration. Third, the type of diarrhea in pellagra, and fourth, we must consider sprue. Certain symptoms are common to all these four forms of diarrhea, namely, stomatitis, anemia and weakness, and loss of weight; and from these symptoms it is difficult to differentiate between these four forms. Of course, in sprue the patient goes on to extreme emaciation, whereas in pernicious anemia or achylia the patient is not infrequently a well nourished individual. Then, again, the picture is confused by the fact that either a gastric or pancreatic achylia may complicate pellagra or may complicate sprue, and it is due to this fact undoubtedly that so much confusion has existed in the establishment of sprue as a clinical entity. There are certain cases of so-called sprue that are absolutely relieved by the exhibition of pancreatic ferment in the diet. Other cases of sprue are relieved by an autogenous streptococcus vaccine. As to the differentiation of these diseases: In achylia gastrica, the absence of the gastric secretion is characteristic; in pancreatic achylia the deficiency of the pancreatic ferment in the duodenal contents or in the stools is characteristic. In pellagra, the skin lesions and the mental symptoms help, whereas in sprue

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examination of the stools is decisive. My personal experience with sprue in Virginia and the neighboring states leads me to confirm the opinion of Dr. Wood and others that so-called tropical sprue is a clinical entity, and that it occurs in the United States in individuals who have never been out of the United States.

DR. JOSEPH H. PRATT, Boston: The patients with tropical sprue that have come to America from the West Indies and from China have had fatty stools. In other words, the diarrhea is a fatty diarrhea. The resemblance of the stools in these cases of sprue to those in complete obstruction of the pancreatic duct is very striking. In three or four cases we did metabolism experiments to determine the amount of fat that was absorbed and the amount of fat that appeared in the feces, and we found that the feces contained so much fat that microscopically they seemed to consist almost entirely of fat. There is no doubt that a great many patients with sprue are coming to this country and cases are developing in the northern states and are unrecognized. Prior to the time that Dr. Wood wrote his first paper there was very little if anything written on the subject of sprue in this country. We should always consider the possibility of sprue in every case of chronic diarrhea with loss of strength and anemia, and examine the stools for fat. The tongue may not be sore at the time the case comes under observation, and the anemia may not be marked but in the cases I have seen, the fatty diarrhea has been the striking feature. I studied very carefully the case of a patient who had not been out of New Hampshire for five years. In that patient the subsequent course and the clinical history were typical of sprue, and if there is one case in the northern states, there must be others.

DR. W. C. ALVAREZ, San Francisco: We see many cases of sprue in San Francisco mainly in people returning from the Orient and India, and I believe that there is something more than the pancreatic lesion present because I have seen pancreatic fatty stools and stools of sprue. In sprue the characteristic thing in all cases I believe is a change of the type of flora in the stools. If you put a sprue patient on a meat diet for a while, he generally improves very markedly. Many patients get well, the bacteria in the stools change, and the stool changes in color. It seems that if it were a pancreatic lesion, the large amount of meat which you feed would not be digested any more than the fat. Take a patient who is comfortable and give him starch, he immediately bloats up with gas if some of those yeasts and moulds are still present. I have seen remarkable cures from the use of vaccine. The strawberry cure is one of the strangest things I have ever seen. Why a man with a violent diarrhea can eat several boxes of strawberries and improve on it

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is a puzzle. You can differentiate sprue from other types of diarrhea by feeding the patient on a box of strawberries.

Dr. Emanuel Libman, New York: Five years ago I saw a case of secondary sprue in New York. The patient had never left New York City. There was complete achylia and disturbance of the reflexes. Up to that time no cases had been called sprue with a color index above 1. These cases are of importance, and we must diagnose between sprue and pernicious anemia. If you analyze the symptomatology of sprue and pernicious anemia, you will see a close relationship. There is a mistaken idea that in pernicious anemia the symptoms are due to anemia, but if you perform transfusion, you can remove the symptoms, the sleeplessness, the poor digestion, etc., and you can raise the functional capacity of the kidneys, but you do not remove the tongue lesions, the achylia, or the spinal cord symptoms. Evidently an anemia is produced by a toxin which has an affinity for the blood forming organs and the spinal cord. In sprue there is a toxin which attacks the gastro-intestinal system and the tongue. As to the spinal cord changes, I do not know what they are, but there is an important difference there. If the toxin of sprue is found, it will give a lead to look out for pernicious anemia.

As to the pancreas: I have seen a few cases of sprue and have been struck by the resemblance to pancreatitis and I have decided to find the exact condition of the pancreatic ferment. Meanwhile, Dr. Thomas R. Brown of Baltimore has reported that in one case of sprue studies of the feces had shown that the pancreatic secretion was entirely missing.

Dr. M. L. Graves, Galveston, Texas: I wish to emphasize the fact that sprue is becoming endemic in this country. Up to a few months ago the only cases of the disease that I had seen in Texas were cases that had been imported from South America and Africa, but recently I saw a case which appears to have originated in Louisiana. An interesting feature of this case is the age of the patient—9½ years. She presents voluminous stools associated with the organism described by Ashford in the Porto Rico cases. This little child presented an epileptoid syndrome which I have not seen in other cases of sprue. The attacks are without a definite aura and occur with great frequency, from three to twenty during the day, associated with great pallor and great sweating. The organisms have also been recovered from the mouth in this particular case. This relationship of epileptoid symptoms to sprue has not yet been established.

Dr. Alfred T. Livingston, Jamestown, N. Y.: On my way to Porto Rico a few years ago I happened to have as companions on the voyage two people affected very intensely with sprue. I became very much interested in them, and before we got to Porto Rico I became a patient myself, hav-

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ing very severe hemorrhages of the bladder, with clots. I was taken to the hospital and operated on, but as soon as I was able after that, I had my wife write to these two people to come to see me because I had conceived an idea that I might be helpful to them. The impression made on me was that the condition was one involving a tremendous amount of fermentation. The stools were so horrible that the husband of this lady could not stay in the house while that operation was going on. So having had a good deal of experience with an antiseptic which I had used in my medical work, without the least suggestion of toxic effect, I thought that if these people could be filled with this antiseptic, immersed in it as it were, they might get relief. One of the persons I met on my trip, a man, did not take my advice. He died within ten days. The other person, a lady, accepted the idea, and made a complete recovery. Other sprue patients have also been relieved by the same antiseptic, betanaphthol.

Dr. Edward J. Wood, Wilmington, N. C.: Dr. Vanderhoof mentioned the occurrence of pellagra and sprue. I have seen that occasionally. The condition which will attract your attention, however, in this event is the superimposed anemia which is caused by the sprue and never by the pellagra. In pellagra the three day test diet shows a normal utilization of both nitrogen and fats even in extreme cases. Dr. Pratt emphasized the existence of fatty diarrhea which, after all, is the essential characteristic of sprue and may be almost counted as a pathognomonic sign. Dr. Alvarez mentioned the value of a strawberry diet as a means of differentiation. I have found the meat diet of Cantlie equally as effective as the strawberry diet. I have seen the color index high, especially just before death, though the English authorities always regard the anemia as secondary, and I believe it to be so. The point of vital importance is the differentiation between pernicious anemia and sprue.

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# HYPERTENSION IN WOMEN

DAVID RIESMAN, M.D.
PHILADELPHIA

The habitual use of the blood pressure instrument has brought me a number of surprises, none greater than the frequency with which hypertension occurs in women. The majority of the cases can be classified in a distinct and definite group having the following features in common:

1. The patients are usually stout, overweight and undersize.
2. They have borne many children.
3. They have neither a history nor any stigmata of syphilis.
4. They are over 45 years of age, the greater number falling between 50 and 60 years.
5. They are practically all constipated and some of them suffer from intestinal indigestion.
6. Up to a certain point they show an amazing tolerance to pressures of high degree.
7. In most instances the heart is enlarged, chiefly to the left.
8. The arteries are soft and even the retinal vessels rarely show any involvement.
9. The kidneys, as far as it is possible to determine, are competent.

The absence of gross renal and arterial changes has led writers to speak of such hypertension as essential, a word of little meaning though quite adequate for concealing our ignorance of the cause of the trouble. A similar hypertension is met with in men; but in them it is less frequent and in general less innocent.

When we come to search for causes, the points that stand out prominently are:

1. Multiple gestations. In nearly every case there have been not only two or three, but often five and

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six children. I have gotten the impression that the worries incident to raising a large family may count for no less than the repeated pregnancies. Inquiry has not elicited a history pointing toward any noteworthy abnormalities either in the pregnancies or in the lying-in periods.

2. Worry. Serious worry is rarely absent: on the one hand, heavy household cares; on the other, special trials and tribulations of the soul.

3. Constipation and intestinal indigestion, especially of the flatulent variety. Just what share to assign to these it is difficult to say. They may play a very important rôle as possible sources of autointoxication. I have, however, seen hypertension of this type in women in whom there was perfect function of the alimentary canal.

4. The menopause. The hypertension begins in such a high percentage of cases during the menopause that this constitutes an unquestionable etiologic factor. The relationship suggests the thought that this form of hypertension in women is based on some endocrine disturbance, not improbably arising in the ovary. Nearly 50 per cent. of the patients were widows.

Whatever the cause may be, whether it is of endocrine origin or some toxic substance made in the intestines or the slow poisoning from a focus of local infection, the effect is an increased vascular tonicity which gradually leads to an actual thickening of the muscular coat of the vessels.

It is interesting to note the symptoms presented by the patients in the early stages of the disease, the inaugural symptoms, to employ a useful phrase of Moynihan's. They are dizziness, ringing in the ears, dyspnea on effort, anginoid pains, palpitation, gaseous distention, and vasomotor disturbances. Though several of these symptoms may be found in the same patient, the disease is often monosymptomatic in the beginning. In a very large proportion of cases the complaints,

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such as brachial neuritis, sciatica, migraine, have no apparent connection with the hypertension.

The chief physical findings are as follows:

The complexion is usually florid; obesity is practically constant; the area of cardiac and of aortic dulness is increased; a systolic murmur at the aortic area, transmitted upward and into the clavicles, and a ringing second aortic sound are common findings. In the later stages a soft systolic murmur can often be heard at the apex. The temporal arteries may be tortuous, but as a rule they are not conspicuous in women. The peripheral arteries are soft, in striking contrast to the blood pressure. An almost constant finding in patients of this type coming to the office is a slight elevation of temperature, between 99 and 100 F.

The average age of the patients was 54 years. The youngest patient was 43, the oldest, 71. The average systolic pressure was 211; the average diastolic pressure, 105; the average pulse pressure, 106; the highest systolic pressure, 310; the highest diastolic pressure, 160, and the highest pulse pressure, 150.

The highest tension, that of 310, was found in a woman, aged 46. Her sole complaint was dizziness. The year previous she had had a uterine hemorrhage. The physician who brought her to me and I myself were completely taken aback when we found the tension so high. From the woman's complaints we had not been led to expect it.

Another patient had a systolic pressure of 290, normal urine, normal eye grounds, and was well and active. She was able to play golf and had practically no complaint save ringing in the ears.

Of course, the type of hypertension I have described is not the only one met with in women. Chronic nephritis with high pressure is fairly common. It may be set down as a general rule that hypertension in women under 35 is practically always nephritic; it

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gives a bad prognosis, much worse than any other form of hypertension. In some of the nephritic cases the arteries are soft and impalpable; in others, they are hard, tense and rigid. Strange to say, the latter condition is more frequent in the younger patients.

There is, furthermore, the arteriosclerotic group in which the etiology, symptoms and course do not differ from those of the same disease in men.

I shall merely mention, without further comment, hypertension in acromegaly and in exophthalmic goiter. I want, however, to say a few words about a closely allied type which I would call non-goitrous thyrotoxic hypertension. This form is of considerable clinical interest; like the one of which I spoke first, it occurs with preponderating frequency in women. The patients are near or past the menopause, are not overfat, sometimes spare, often single. They complain chiefly of palpitation and headache; are emotional, and have a tendency to sweating. The heart is rapid, even to the point of tachycardia; the hands are tremulous. There is often a von Graefe sign, but no exophthalmus and no goiter or other tangible abnormality of the thyroid gland. The systolic pressure ranges from 150 to 210, with a diastolic pressure of from 90 to 120. While there is no positive proof of a thyroid cause, and while the patients are nearly all beyond the age at which hyperthyroidism usually appears, the symptoms nevertheless closely resemble those of a thyrotoxicosis. To be sure, the condition may be pluriglandular in origin. It is apparently not renal or arteriosclerotic. A point of interest and in a sense contributory evidence of thyroid origin is the fact that the iodids do not benefit but rather harm these patients. The thyrotoxic form is more incapacitating than the first variety of which I have spoken.

With regard to prognosis, the first type, the so-called essential hypertension, is in a sense benign; even high pressures are well tolerated for many years, as I

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have pointed out in earlier papers on this subject.[1] Hence, it is both unwise and unnecessary to alarm these patients. Many have been unduly frightened by being told that they had very high blood pressure. Though the physician, in dealing with them, may well be optimistic, nevertheless, for the patients' sake it is important that he keep them under surveillance on account of the possible catastrophes that lie in wait for them. Three major accidents may happen:

1. Angina pectoris, which is much more frequent in women than is believed, and is especially common in the so-called essential type of hypertension. It is probably due to an extension of aortitis to the coronary arteries.

2. Apoplexy with hemiplegia, which latter in the majority of my cases has been left-sided.

3. Decompensation, a late stage of the disease resulting from myocardial exhaustion.

### TREATMENT

In this, as in all chronic diseases not definitely infectious in character, our powers of cure are very limited. That being so, the great desideratum is to find means of prevention. To this end we must learn to recognize hypertension in the making and then to remove such removable factors as are concerned in its production. As there may be nothing in the symptomatology in the early stages to attract attention to the circulatory system, there is but one way to discover incipient stages—by routine blood pressure examination and careful determination of the size of the heart. A comparison of records made at intervals would then show us in which patients the pressure was on the upgrade.

In the treatment of the established condition, harm may readily be done by too much interference. Just as we have gotten away from using digitalis on the mere

1. Riesman, David: Penn. M. J. **18**: 193-202 (Dec.) 1914; Am. J. Obst. **71**: 428-133 (March) 1915; Am. J. M. Sc. **165**: 487-494 (April) 1913.

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discovery of a murmur when the valvular defect is compensated, so we should not try to lower the blood pressure to any great degree in patients in whom the hypertension is well borne—in whom it is adequately compensated. If the patients know that they have high blood pressure, one may honestly assure them that the dangers of this symptom have been overemphasized.

Regulation of the diet is necessary, more often in the direction of quantity than of quality. I have found this class of patients as a rule to be heavy eaters, and have come to feel that they do better on a restricted diet, especially on one that is largely lactovegetarian. A lamb chop, a little chicken and fresh fish are permissible. Rest, at times a semirest-cure, is desirable in many instances. In some persons, however, graduated exercise, walking and moderate golf playing may safely be advised.

With regard to drugs, I am not in the habit of using them as a matter of routine. The nitrites are not indicated in patients who do not suffer from their hypertension. Though high-geared, the circulation may be well maintained and vasodilators only serve to upset the equilibrium. The iodids may be used in the essential type of hypertension in small doses over a long period. Lately, I have secured apparently striking results from the use of corpus luteum extracts. The pressures were lowered without any untoward effects, and the subjective symptoms were markedly improved.

Baths and electricity may at times be employed with benefit. When the cases approach the danger line, nothing is so valuable as venesection; when this cannot be done, then leeching from the mastoid process is a very good substitute.

In the thyrotoxic cases, rest is of the greatest importance. Patients of this type should take their breakfast in bed and lie down for an hour or an hour and a half after the noon meal. Tea and coffee should be forbidden. Medicinally, the bromids, at times, with

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small doses of tincture of veratrum, seem to do good. I may add that in the dizziness of hypertension, tincture of veratrum has often given me very satisfactory results.

The patients of all types of hypertension should be urged not to worry, but the mere injunction "not to worry" carries small weight. It is necessary for the physician to analyze with judgment and with great patience the worry-breeding conditions of the person's life and to convert his patient, if possible, to a more philosophic attitude. By considering all the details of the patient's daily life, one may often point out definite ways of easing the strain.

## ABSTRACT OF DISCUSSION

Dr. Joseph H. Pratt, Boston: Dr. Riesman has done well to emphasize the tolerance with which high blood pressure is borne for a number of years by certain patients, especially by women of middle age and above who exhibit no signs of renal disease. The cases I have studied of high blood pressure with normal renal function may be divided into three groups: 1. Vasomotor neurosis with transitory hypertension. The blood pressure is unusually labile. Slight emotion produces a marked rise. These cases are properly regarded as neurasthenia with vasomotor disturbances. The rise in blood pressure is attributed to vasomotor spasm. Other symptoms due to spasm of the vessels are frequent 2. Primary permanent hypertonia. Elevated blood pressure may be the only sign of disease and there may be no subjective symptoms. Most of the patients at the time they come under observation have enlargement of the heart. I have seen cases, however, in which there was no evidence of cardiac hypertrophy. Dr. O'Hare, in an unpublished study, has found in hypertonia without renal disease a marked increase in the blood pressure after an injection of epinephrin. This indicates a state of increased tone in the sympathetic nervous system. 3. Localized arteriosclerosis with hypertension. In a large number of cases in this group of localized arteriosclerosis there is evidence of cardiosclerosis. As Dr. Riesman states that in his cases there is an increased area of aortic dulness and a systolic murmur at the base, as well as an enlargement of the heart, it would seem as if they should be placed here. Is the hypertonia primary and the heart hypertrophy secondary, or are the physical signs of involvement of the heart found in the earliest stage? Orthodiagrams and teleradiograms of the heart taken at

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intervals beginning, if possible, before hypertonia developed and continued over a series of years, would yield information of value. As evidence of emotional instability of the blood pressure is such a marked feature in essential hypertonia, it may be that some of the cases with transitory elevation of blood pressure tend to pass into the second group characterized by a persistent hypertonia. Cases of this type sooner or later exhibit definite signs of localized arteriosclerosis and then belong in the third group. This progression from one group to the next indicates that we may be dealing with different stages of the same disease process which begins with a transitory spasm of the vessels and ends with widespread arteriosclerosis.

Dr. Louis Faugeres Bishop, New York: The whole question of the disposition of these very high blood pressure people is so important that it is well to emphasize some practical points in their care. What shall we do with these people so that they will live for a long time? For the last twelve years I have done nothing but treat blood vessel and heart cases and refer now to pressures of from 210 to 260. I have taken care of many of these persons for long periods of time. And there are certain things that are absolutely the best for them. In the first place, I would select the best remedy of all as outdoor exercise. If you can get these women with very high blood pressures and all the nervous symptoms and everything else that goes with them to take a long walk every day, you have won half the battle in taking care of them, because exercise is the best heart stimulant in the world; it is the best lung stimulant; it is the best kidney stimulant; it is the best antidote to worry. Next to that I would place diet. The most essential element in the treatment of this group is the exclusion of eggs from the diet. Some are sensitive to eggs, but not all of them. But, as a matter of strategy, the exclusion of eggs is essential. If you exclude eggs from the cooking, you exclude a great deal of the protein. In our present economic status eggs are very objectionable, because of poor quality. Of course, excess of meat should be avoided. The third important element is care of the intestinal tract. Most of these people are laxative fiends. They have been taking laxatives all their lives and they suffer from autointoxication which comes to people who abuse laxatives. Castor oil is the best of all. I give them a full dose on alternate nights for a week, then skip a week, then two weeks, then three weeks and then advise a full dose once a month as long as they live.

Dr. J. M. Anders, Philadelphia: The fact is recognized that chronic hypertension is of the greatest aid in the recognition of chronic nephritis. It is important to recollect, however, that this symptom has not quite the same value in

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the female as in the male, for the reason that the female hypertension of vasomotor toxic origin is more frequent than in the male. We meet with many cases of chronic hypertension at the time of the menopause due to endocrine causes, and most of them are quite independent of the group so well described by Dr. Riesman. Then again in true exophthalmic goiter, and also in the cases which have been referred to by Dr. Riesman, as "thyrotoxic symptoms without thyroid disease or exophthalmos," we have instances of chronic toxic hypertension under conditions that do not lead to chronic nephritis as a rule. There is a form of chronic myocardial disease, with which chronic hypertension is often associated, in which there is more or less myocardial insufficiency, but the blood pressure is abnormally high. There is a vasomotor compensatory mechanism operative in such cases; this is practically proved by the fact that as you strengthen the myocardium the blood pressure falls. This particular variety of myocardial disease is more common in the female than in the male. These subjects are often obese; they have reached middle life and commonly anemia of the secondary type is found in association; and both the obesity and the anemia must be taken into account in the treatment since both are causally related to the myocardial condition. I have seen several cases which belong to the group first described by Dr. Riesman in which cardiac hypertrophy with hypertension, and the basic systolic murmur, angina and elevation of temperature were the principal symptoms. To the causes which he mentioned of this condition, menopause and mental worry, I would add overeating with insufficient systematic muscular exercise. The murmur is not due to stenosis at the aortic orifice but doubtless to a roughening of the valve segments or of the intima of the aorta. The slight fever is most probably due to an associated aortitis, and if we accept Allbutt's view, the aortitis accounts for the angina pectoris. Of the two cases I have seen which belong to this group one patient died of angina pectoris and the other has recently had her first attack. In regard to the treatment of this condition, I feel that the two most important elements are: first, proper restriction of the diet, particularly the fat-forming elements, and, second, systematic physical exercise, as Dr. Bishop has pointed out, provided the arterial changes referable to the aorta are not too far advanced. If there is extension of the aortitis to the coronaries with marked arrhythmia, then, of course, this question of systematic physical exercise must be taken up with great care and caution.

Dr. David Riesman, Philadelphia: What Dr. Pratt says about sclerosis of the aorta is true. However, though there may be arterial changes elsewhere, the peripheral arteries are usually so soft and pliable that unless one is careful to take blood pressures, the circulation will be considered

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normal. The patients come to us when they are in the state of hypertension. To detect the earliest stages we must, as Sir James Mackenzie suggests, make complete examinations and take full histories of all patients, even if the complaints are trivial. Reviewing the histories of those cases that develop hypertension, it will eventually be possible to know the beginnings of the disease. Something may, perhaps, be learned from the periodic examinations inaugurated by some insurance companies. Dr. Bishop's observations are most interesting. If I lived in New York I should probably agree with him regarding eggs. I am, however, not in favor of a high protein diet in hypertension cases. Patients have told me that they felt better on a diet largely lactovegetarian and they do not want to return to a meat diet. But in the last analysis it is probably more important to reduce quantity than to alter quality. What Dr. Anders says about taking blood pressures, in order to recognize nephritis, is most important, as the blood pressure often gives us a better idea of the duration and gravity of the disease than the urine. Anemia is sometimes an important feature and patients are frequently helped by treatment directed against the state of the blood. Regarding exercise, I believe there are periods when it is better not to allow it. One must be governed by the response of the circulation to physical activity. In many cases golf undoubtedly proves beneficial, perhaps as much by reason of the mental relaxation as for any other reason.

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# THE FUNCTIONS OF THE INTERCOSTAL MUSCLES

## FURTHER OBSERVATIONS

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In two former publications[1] I sought to show how observations on the respiratory movements of the costal margins give us information which enables one to gain definite conceptions about the conformation of the diaphragm. Briefly stated, the intercostal muscles and the diaphragm are antagonists. The minus barometric pressure produced in the pleural cavity during inspiration is due entirely to the excursion of the ribs and the diaphragm. The costal excursion increases the capacity of the thorax in its anteroposterior and transverse diameters, and the phrenic excursion alone increases the longitudinal diameter of the thorax. If all these muscles are activated at the same moment, it is clear that to accomplish an excursion of the ribs and diaphragm, the intercostal muscles and the diaphragm must each reciprocally overcome the minus pressure created by the other. The diaphragm must overcome the minus pressure produced by excursion of the ribs before there can be phrenic excursion, and the intercostals must overcome the minus pressure produced by excursion of the diaphragm before there can be any costal excursion. The lower six intercostal muscles and the diaphragm are also antagonists in controlling the movements of the costal borders.

1. Hoover, C. F.: The Functions of the Diaphragm, Arch. Int. Med. **12**: 214 (Aug. 1913; Diagnostic Signs From the Scaleni, ibid. **20**: 701 (Nov.) 1917.

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Unopposed by phrenic action, the intercostal muscles will cause the entire costal margin to move away from the median line and, unopposed by the intercostals, the diaphragm will draw the entire costal margin toward the median line.

If the diaphragm is paralyzed, or its costal attachments are cut away, the outward movement of the costal margin will be greatly exaggerated. If the intercostal muscles are paralyzed, the costal margins will move toward the median line during inspiration.

The intercostal muscles and the diaphragm are antagonists in determining the direction in which the costal margins will move during inspiration, but the direction in which the costal margins may move does not in any way express the intrapleural pressure during inspiration. Nor does the direction in which the costal margins move give expression to the inspiratory excursion of the lung. In other words, there may be adequate (not normal) inspiratory enlargement of the lungs in all three dimensions whether the costal borders move in a median or a lateral direction.

Former interpretations of respiratory movements of the costal borders were based on the experiments and interpretation of Duchenne, which considered only the factor of increased intra-abdominal pressure during descent of the diaphragm as the source for lateral movement of the costal borders.

### CONTROL OVER COSTAL MARGINS

I proved by clinical observations and animal experiment that the balance of control over the costal margins depended on the arch of the diaphragm.

In human beings the intercostal muscles have a small margin of superiority over the diaphragm in determining the direction in which the costal margins will move. The more nearly the curve of all, or any part of, the diaphragm approximates a plane, the greater the mechanical advantage will that part of

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the diaphragm have in control of the costal margin to which it is attached. The anteromedian portion of the diaphragm is less arched than its lateral-posterior portion; consequently less depression of the anteromedian portion is required to give that part of the diaphragm mastery over the costal margin to which it is attached than is required in the case of the lateral-posterior part of the diaphragm.

The phrenic muscle fibers which underlie the pericardial sac are assigned to the costal margins from the subcostal angle to the eighth cartilage on the costal margin. When all the chambers of the heart are enlarged, as in decompensated hearts from myocardial or mitral disease, and when the pericardial sac is enlarged, then the anteromedian or sternocostal part of the diaphragm is depressed, and, as a consequence, the examiner will see a symmetrical narrowing of the subcostal margin during inspiration; but beyond the eighth costal cartilage the costal margins are seen to move away from the median line during inspiration.

The balance of control over the costal margin is so nicely adjusted, and depends so wholly on the curvature of the diaphragm, that any asymmetry of phrenic curve to the two sides of the median line will make itself apparent in asymmetrical movement of the costal margins.

If the left ventricle is enlarged and the right ventricle and right auricle are not greatly enlarged, the left costal margin (to the eighth rib) will move less in an outward direction than the corresponding right side, and the left side may move toward the median line if there is sufficient flattening of the diaphragm. If the right ventricle and right auricle are enlarged, and the left ventricle is not enlarged, then the right costal margin, as far as the eighth rib, will be restricted in its outward movement and may, indeed, move toward the median line with inspiration. So the direction of movement of the subcostal angle and the

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symmetry, or asymmetry, of movement of the two sides may be employed to interpret the arch of that part of the diaphragm underlying the pericardial sac; and, inferentially, we gain valuable evidence concerning the size of the pericardial sac and also evidences of the size of the left ventricle, right ventricle and right auricle.

### SYMMETRY OF MOVEMENT

The symmetry of movement of the entire costal borders gives evidence of the symmetry of the right and left vaults of the diaphragm. Any condition which will increase the arch of one side of the diaphragm, such as enlargement of the liver or subphrenic abscess, will cause the costal margins to move farther laterally, and more promptly, than the costal margin of the other side. When the right thorax, in one instance, was filled with pus which followed the rupture of a large subphrenic abscess through the diaphragm, the right costal margin moved farther away from the median line during inspiration than did the left margin where the lung was perfectly normal.

The movement of the costal borders is a reliable index of the elevation or depression of the arch of the diaphragm.

When the lung is enlarged by atrophic emphysema or by hypertonus of the bronchiolar musculature, the costal margins are drawn toward the median line during inspiration.

In cases of pneumothorax and empyema, when the under surface of the diaphragm is convexed, the costal margin moves away from the median line during inspiration. When air is released, or fluid is aspirated so that the leaf of the diaphragm is no longer convex on its under surface, the costal margin will move toward the median line when the diaphragm is activated, although under such conditions there can be no phrenic excursion.

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The movement of the costal margin is determined by the activation and arch of the diaphragm and is not dependent on the excursion of the diaphragm.

The failure to differentiate between evidences of excursion and evidences of activation of the phrenic leaf, and the failure to appreciate the mechanical significance of the diaphragmatic arch have led to misinterpretation of the action of the diaphragm.

Study of the respiratory movements of the costal margins gives valuable information concerning the conformation of the entire diaphragm, and it also enables us to recognize any disturbance of balance between the intercostals and diaphragm when the existing impairment is on the part of the intercostal muscles.

In former years, when evidences of excursion were wanting, the diaphragm was supposed to be paralyzed, and no significance was attached to the direction or degree of excursion of the costal margins.

The excursion of the abdominal wall is a measure of the excursion of the diaphragm, but the excursion of the costal margins simply indicates the resultant of the two forces which are evidenced in the direction and degree of excursion of the costal margin. These two forces are derived from phrenic and intercostal muscle activation and must not be confused with evidences of excursion of the diaphragm.

### PHRENIC AND INTERCOSTAL MUSCLES

The more nearly the phrenic muscle approximates a plane, the greater mechanical advantage will it have in procuring mastery of the costal border; and the more curved the plane of the diaphragm, the less evidence will there be of its activation. It matters not whether the diaphragm is convexed downward, as in empyema and pneumothorax, or convexed upward, as in hepatic enlargement or subphrenic abscess. Although the diaphragm moves upward (in some cases

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of pneumothorax and empyema) during inspiration, the costal margins of the affected side will move outward during inspiration. This experience, which can be seen plainly with the fluoroscope in patients who have a pneumothorax under positive pressure, very definitely disposes of the old interpretation of inspiratory spreading of the hypochondria, which was based on Duchenne's experiments.

Movement of the ribs and the degree and direction of movement of the costal borders give valuable aid in detecting evidences of paresis of intercostal muscles. The costal excursion very faithfully mirrors the excursion of the underlying lung. This is of great importance in physical examinations, for it enables the examiner to detect regional modifications of pulmonary excursion.

By physical examination the physician should form very definite ideas of the size, density and extensibility of the lungs. Indeed (with the exception of a few adventitious signs), all of our methods of pulmonary examination are directed toward an estimate of these three attributes of the lung. Of the three attributes, namely, size, density and extensibility, the last is the most exquisitely responsive to pathologic changes. Extensibility is the first physical attribute of the lung which is modified in all diseases of the bronchi, lung tissue and the pleurae. It requires only a few moments to palpate the respiratory excursion of all the ribs and determine the character of excursion of the costal margins. When these observations are critically performed, the examiner will have formed a definite conception of the conformation of the diaphragm, and also a clear idea of the extensibility of the upper and lower lobes of the lungs.

Although paralysis of the intercostal muscles will diminish the vital capacity of the lungs in direct proportion to the extent of the lesion, all paralyses are well tolerated excepting one combination, and that is a

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paralysis of the upper half of the intercostals when the remaining lower half of the intercostal muscles are active.

## REPORT OF CASES

CASE 1.—A plump, healthy looking boy, aged 2 years, was brought to the hospital on account of what seemed to be paroxysms of suffocation. Stenosis of the upper air passages was suspected.

The child lay very comfortably on his back in bed and exhibited no signs of distress unless he was engaged in play, and laughed or cried. Any activity which was attended with an increase of respiration was attended with pronounced cyanosis. Dyspnea and cyanosis were so pronounced it frequently seemed that suffocation was imminent.

Cyanosis and respiratory discomfort increased in a vicious cycle as any incentive to deep breathing was employed. The more deeply and rapidly the child tried to breathe, the worse it breathed. There was no prolongation of either expiratory or inspiratory phase. It was also observed that during these violent inspiratory efforts, the upper thorax was violently retracted and the lower thorax was violently distended, with exaggerated spreading of the hypochondria and protrusion of the abdomen. There was a genuine see-saw between the upper and lower thorax. The scaleni were intact, as was evidenced by fixation of the first rib of each side and a want of descent of the manubrium during inspiration.

During an inspiratory effort the ribs on both sides, from the second to the sixth inclusive, were strongly retracted. The seventh rib (on both sides) was stationary. From the eighth rib to the twelfth (inclusive) there was an exaggerated excursion in a normal direction.

It was clearly evident in this case that there was a marked diminution in volume of the upper lung and exaggerated distension of the lower lung during a violent inspiratory effort; the lower lung was, therefore, distended in part by air which was displaced from the upper lung during inspiration; so the patient was ventilating his lower lung in large part by alveolar air from the upper lung, and during expiration, alveolar air from the lower lung was driven into the upper lung. There was a reciprocal expansion and contraction between the upper and lower lung. During strong inspiratory efforts, alveolar air was shunted back and forth between the upper and lower portions of both lungs. Tranquil breathing was attended with adequate ventilation, but forced breathing developed a vicious reciprocal contraction and expansion between the upper and lower parts of both lungs, which caused intense air hunger and cyanosis.

There were no physical signs to indicate any disease of the bronchial tract, or lung parenchyma or pleurae. A

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roentgenogram revealed marked signs of tuberculous disease of the bodies of the upper six dorsal vertebrae, in other respects revealing a perfectly normal thorax.

There were no other neurologic signs obtainable which would indicate any disease of the spinal cord or the intercostal nerves. The respiratory distress and inspiratory retraction of the second to the sixth ribs (inclusive), associated with the disease of the vertebrae revealed by the roentgen ray, seemed, however, to justify a diagnosis of motor paralysis of the intercostal muscles, which normally accomplish the ventilation of the upper parts of the lungs.

The vicious cycle of rebreathing established between the upper and lower lung was due to the flexibility of the child's ribs, as well as to the motor paresis of the upper intercostal muscles. Had the same extent of motor paralysis occurred in an older person, with more resistant ribs, there would probably have been little or no respiratory distress. This is the only occasion in which I have seen paralysis of the upper intercostals when the lower intercostals were active, and is the only intercostal paralysis which will cause reciprocal rebreathing between two regions of the lungs. I have frequently seen paralysis of the lower intercostal muscles when the upper muscles were intact and, on one occasion, motor paralysis of all the intercostal muscles with scaleni and diaphragm intact; but the patients had no respiratory distress, because there was no rebreathing between different parts of the lung.

Paralysis of all intercostal muscles on one side is also well tolerated.

CASE 2.—A girl, aged about 6 years, was brought to the hospital directly after she had fallen to the ground from a ladder on which she was climbing. The resident physician palpated what he interpreted as the body of the third cervical vertebra projecting into the posterior pharyngeal wall. The projecting mass was pushed back into position. It was then discovered that the child had motor paralysis of the left upper and lower extremities, and it was also observed that the left side of the thorax did not move with respiration. A lumbar puncture revealed a large amount of free blood in the subarachnoid space. There was complete paralysis of all the intercostals of the left side of the thorax, and there was no demonstrable evidence of any activation or excursion of the left side of the diaphragm. All the pulmonary ventilation was accomplished by the intercostals and diaphragm of the right side.

The child was not cyanotic, she had no air hunger, and there was no inspiratory retraction of the left thorax. The entire left thorax was completely immobilized. There must have been some ventilation of the left lung because there would certainly have been cyanosis at least, had all the blood passing through the left pulmonary veins been unre-

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spired. A very moderate increase in respiratory excursion of the normal right lung would have compensated for an increase of carbon dioxid contributed to the aortic blood by unrespired blood from the left lung; but superventilation of the right lung could not prevent cyanosis from appearing under such circumstances. We would have seen the same degree of cyanosis that appears in fibrinous pneumonia of the entire left lung. Were there no ventilation of the left lung under such conditions as presented in this child, then the only area for the escape of carbon dioxid and absorption of oxygen for the left lung would have been the cross section of the left bronchus.

When we consider the estimated area of the total respiratory membrane of an adult lung to be 90 square meters, it is apparent that although a bronchus to a lung may be open, it offers a negligible area for transpiration of gases. In the case of this child, with all the intercostal muscles and diaphragm of the left side paralyzed, the only manner in which anoxemia could have been prevented would have been through some respiratory excursion of the mediastinum, for there certainly was no respiratory excursion in a longitudinal or anteroposterior diameter. There must have been some respiratory movement in the third dimension. With the disturbance of equilibrium between the right and left pleural cavities which attended intercostal and pleural excursion of the right side, the mediastinal structures moved toward the right. In this manner the transverse diameter of the left pleural cavity was enlarged during inspiration and diminished during expiration. There was therefore no rebreathing between any two portions of the lung.

To summarize, paralysis of all the intercostal muscles, of both sides, or of one side, or the lower half of both sides, or the lower half of one side, produces no respiratory distress nor cyanosis when the patient is at rest.

Paralysis of the upper half of the intercostals of both sides, however, produces severe respiratory distress when the ribs are sufficiently flexible to admit of reciprocal expansion and contraction between the upper and lower parts of the lungs so as to cause rebreathing between the upper and lower lung.

Although this manner of intercostal paralysis is the only kind which will per se produce suffocative symptoms, intercostal paralysis of a moderate extent will diminish the vital capacity of the lung.

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CASE 3.—A man suffering from syringomyelia of the cervical and dorsal cord had very moderate emphysema which was not sufficient to cause flattening of the diaphragm; but the pulmonary volume was enlarged so that the lower border of the lung stood one intercostal space lower than is normally found. The striking feature about this patient was the very small margin of cardiorespiratory reserve, which was disproportionately small for the very moderate emphysema. Physical examination and the roentgen ray both failed to detect any signs of lung or heart disease which could in any way explain the small vital capacity of the lung, which was only 1,100 c.c.

Pulmonary emphysema which would reduce the vital capacity to so small an amount must be a severe emphysema, and this man did not have severe emphysema, nor was there any evidence of cardiac enlargement, or stasis in the pulmonary circulation, or other disease of the bronchi, or lung parenchyma or pleurae. The striking disparity between the moderate emphysema and great reduction in the vital capacity could be accounted for only by evidences of paralysis of the intercostal muscles from the third to the seventh rib (inclusive) of the left side. The left costal border, from the subcostal angle to the eighth rib, was drawn toward the median line during inspiration. The left costal margin below the eighth rib and the entire right costal margin moved laterally during inspiration. When the ribs of the left side were palpated successively from above downward, all could be felt to move normally excepting the fourth to the eighth ribs, which, on palpation, revealed inspiratory retraction.

There was no evidence of disease of the underlying lung, and there was not merely a want of normal excursion of these five ribs, but there was a genuine retraction of the ribs during inspiration.

The retraction of the costal border of the left side, from the angle to the eighth costal cartilage, was explained by the normal diaphragm in this region being unopposed by intercostal muscles. Further confirmation of this interpretation of the small vital capacity was found in evidences of marked paresis of the abdominal muscles of the left side. During expiration the abdominal wall was seen to be rotated on a longitudinal axis; the median line was drawn toward the right side during forced expiration, and the umbilicus moved upward and toward the right when the patient coughed.

During the act of coughing, one could palpate an equal bursting tension over the left and right sides of the abdomen; but the abdominal wall to the left of the median line was clearly seen to be protruded, while the right side was retracted. To the left of the median line the abdominal wall was passively distended; but to the right of the median

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line the abdominal wall was actively contracted, when the patient coughed or made a forced expiration.

An analysis of this patient's peculiar respiratory limitations and movements became quite clear when the movements of the costal margin were employed to study the balance of power between the diaphragm and the intercostal muscles; but aside from this point of view, his respiratory movements became a hopeless tangle.

### INTERCOSTAL MUSCLES AND DIAPHRAGM

In several cases of syphilis of the spinal cord it has been possible to demonstrate paralysis of several groups of intercostal muscles by observing the respiratory movements of the entire costal margin. When the diaphragm is unopposed by intercostal muscular action, the costal border will be drawn toward the median line during inspiration; but the retraction of the costal border does not prove that there is impaired muscular power of the intercostal muscles—the intercostal muscles may be normal and the costal margin be retracted because the flattening of that particular part of the diaphragm gives the phrenic muscle mastery over the costal end. When retraction of the costal margin is an evidence of paresis or paralysis of the intercostal muscle, then the involved rib will be seen to retract throughout its course, whereas, the anterior end of the rib will be retracted, and the rib in its course will be normally elevated, when flattening of the phrenic arch is responsible for retraction of the costal border.

In studying the respiratory excursion of the ribs, one must differentiate between the significance of excursion of the costal ends, namely, the costal border, and excursion of the arcs of the ribs. The direction of the excursion of the ends of the ribs (costal border) is quite independent of excursion of the diaphragm and excursion of the arc of the rib. The costal border (rib ends) will move either toward the median line, or laterally, according as the pull of the diaphragm or of the intercostal muscle may have the

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mastery of the ends of the ribs. When the intercostal muscles are paretic or paralyzed, the costal border formed by the ends of the affected ribs will be drawn toward the median line during inspiration, and the arc of the affected ribs will also be retracted during inspiration.

When the diaphragm is flattened and the intercostal muscles are normally activated, then the costal border will be drawn toward the median line during inspiration; but the course of the rib will move in the normal "bucket handle" arc.

Careful study of the respiratory excursions of the arcs of the ribs and of the costal border gives very valuable information on the innervation of the diaphragm and the intercostal muscles. By this study we are also enabled to visualize the conformation of the leaves of the diaphragm, and by such observations we also gain valuable information about the extensibility of the lung.

## CONCLUSION

Costal movements accurately mirror the movements of the underlying lung.

When the respiratory excursions of the thorax are studied in this manner, the lung and its bounding walls take on an entirely new significance.

The lung will be appreciated as a very lively organ, which has a motility quite different from the popular conception of an elastic bag which is alternately expanded and contracted en masse in obedience to changes of barometric pressure within the pleural cavity.

1903 East Seventy-Fifth Street.

## ABSTRACT OF DISCUSSION

Dr. Henry Sewall, Denver: Dr. Hoover has opened what is practically a new field in the clinical physiology of respiration. Since Hutchinson, in 1846, showed that the lung ventilation could be separated into four compartments, namely, supplemental, residual, complemental and tidal air,

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we have learned next to nothing, we have done next to nothing. Many years ago I published a paper on the relation of diaphragmatic to costal respiration. I learned enough to see that there is a tremendous field for research, one which is rich in possibilities. I am afraid I did not uncover it. Dr. Hoover did not speak of the relation of respiratory movement to the blood flow through the lungs. That is a matter of supreme importance. The muscles which increase the capacity of the chest play the part of a suction pump and the muscles which decrease the capacity of the abdomen act as a force pump to send the blood to the heart and lungs. Much functional disability depends on inadequacy of these secondary aids to the circulation. Dr. Hoover referred to the fact that, under certain conditions, air is pumped by respiratory movement from one part of the lungs to another. This is a very interesting fact. You can demonstrate certain practical conclusions by the use of this method voluntarily. Hold your breath as long as you can, with glottis closed—say, one minute. When you can hold the breath no longer, simultaneously contract your chest and expand your abdomen and vice versa, and you churn the air back and forth in your lungs and you can hold the breath for, say, a minute and a half. As the surgeons are appreciating the fact that there is a field for thoracic surgery, the clinicians must realize that there is a mechanics of respiration which they have always avoided because they lack mechanical training. It is just as important as the study of the signs of appendicitis.

Dr. Charles L. Minor, Asheville, N. C.: As a result of many physical examinations I have come to believe that the presence of a very small focus of infection in even the upper portion of the lung will almost always manifest itself quite clearly by limitation of motion of the base of that lung, either in front or behind, and that such a limitation will often antedate a narrowing of the pulmonary isthmus, called Kroenig's sign, and such a limitation of motion of the base, unless it can be easily explained on some other ground, is, to my mind, a strong diagnostic point in favor of the existence of a focus of tuberculosis somewhere above in the lung. In determining such a limitation, I find percussion distinctly more valuable than the use of the fluoroscope. The latter shows the motion of the dome of the diaphragm but it does not show retardation of motion in the complemental space. Often I find that such a limitation in a case that is otherwise suspicious, but not more, will go far to clinching a diagnosis, so that I would note that when the focus is nearest the back, the limitation will be chiefly in the back, when nearer the front of the lung, it will be chiefly in the front. After Dr. Hoover's remarks on the motion of the second or third rib, I regret that I have not had at my disposal Ransom's stethometer, an instrument by which one can measure accurately the mobility of any individual rib. This

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instrument is fully described in his work on stethometry. In recent years, with the advent of the roentgen ray and other measures, physical diagnosis has been too much neglected. It should always be remembered that in the diagnosis of tuberculosis, little things are of infinite value, and none of the various steps should be omitted since very frequently the summation of several small points will bring a diagnosis, where had they been neglected in our haste at examination, a diagnosis would be impossible. This is why neither palpation nor mensuration, which in themselves are not very valuable, should never be omitted. Sometimes they will give a little extra light which is of infinite value. The only safe rule in a careful physical diagnosis is to omit none of the recognized measures.

Dr. Chas. F. Hoover, Cleveland: The point Dr. Sewall mentions is one which concerns the inspiratory impulse in the presence of varying amounts of oxygen. The impulse to breathe may be satisfied for a brief period by relaxing the inspiratory muscles for a moment, although no air is taken into the lung. This gives momentary relief from the painful impulse to breathe. When the lungs are filled with pure oxygen a normal person can hold his breath for three minutes and thirty seconds. The expired air will contain as much as 9.3 per cent. $CO_2$. When the lung is filled with atmospheric air the same person can hold his breath only one minute and twenty-five seconds and the expired air will then contain only 6.3 per cent. $CO_2$. The varying tolerance of $CO_2$ in the blood with different concentrations of oxygen is well known but thus far there is no satisfactory explanation for the phenomenon. Descent of the lung into the pleural sinus is not a guide to the increased volume of the lung. The lung may permanently fill the pleural sinus on days when there is air hunger, cyanosis and high $CO_2$ content of the alveolar air and also on days when there is no air hunger and no cyanosis and a normal $CO_2$ content of the alveolar air. The only way in which the increased emphysema can be recognized is to observe the movements of the costal borders, which betrays the varying degrees of flattening of the diaphragm.

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# PERICARDITIS AS A COMPLICATION IN PNEUMONIA

## BASED ON THREE HUNDRED NECROPSIES

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Records of 300 necropsies on pneumonia patients from the Medical Service of the U. S. Army base hospital at Fort Riley, Kan., are available for the purpose of this study. Among this number, pericarditis was found to have occurred in seventy-two, or 24 per cent. The types of pericarditis, as a complication in pneumonia encountered at necropsy, are shown in Table 1.

TABLE 1.—TYPES OF PERICARDITIS IN PNEUMONIA

| | Number | Per Cent. |
|---|---|---|
| Acute serofibrinous pericarditis | 14 | 19.4 |
| Acute purulent pericarditis | 44 | 61.1 |
| Subacute fibrinoplastic and purulent pericarditis ("shaggy heart") | 14 | 19.4 |
| Total | 72 | |

In addition, hydropericardium, without apparent inflammatory change, had occurred in twelve instances, or 4 per cent. Empyema was present in sixty-three of the seventy-two instances of pericarditis, or 87.5 per cent.

### ACUTE SEROFIBRINOUS PERICARDITIS

In approximately 5 per cent. of the pneumonia patients on whom necropsy was performed, acute serofibrinous pericarditis had occurred. Among the fourteen instances, lobar pneumonia had occurred in eleven, and bilateral bronchopneumonia had occurred in three. This form of pericarditis was characterized by the presence of a flaky pericardial fluid, which was cloudy in appearance and which contained strings of fibrin adherent to the epicardium and pericardium. The quantity of fluid varied from 250 to 1,000 c.c.

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### ACUTE PURULENT PERICARDITIS

In approximately 15 per cent. of the pneumonia patients on whom necropsy was performed, acute purulent pericarditis had occurred. Among the forty-four instances, lobar pneumonia had occurred in thirty, bronchopneumonia had occurred in eleven, while a combined form of lobar and bronchopneumonia had occurred in three. The amount of pus in the pericardium varied from 100 to 1,000 c.c.; the average in twenty-six instances in which the amount was recorded was 350 c.c.

### SUBACUTE FIBRINOPLASTIC AND PURULENT PERICARDITIS

In approximately 5 per cent. of the pneumonia patients on whom necropsy was performed, the subacute form of pericarditis, characterized by a fibrinoplastic or villose and purulent exudate, had occurred, leading to the pathologic picture of the "shaggy heart" or cor villosum. Among the fourteen instances, lobar pneumonia had occurred in eight, and bronchopneumonia in six. The pericardium in all instances was lined by a partly organized villose exudate adherent to the epicardium. The amount of free purulent fluid usually associated with the fibrinoplastic exudate varied from 15 c.c. to 600 c.c., the average being about 250 c.c.

In Table 2 is shown the extent of the associated pneumonia incident to the three types of pericarditis.

TABLE 2.—PERICARDITIS AND THE EXTENT OF THE ASSOCIATED PNEUMONIA

| | | Pneumonia | | | | | |
|---|---|---|---|---|---|---|---|
| | | Both Lungs | | Right Lung | | Left Lung | |
| | No. | No. | Per Cent. | No. | Per Cent. | No. | Per Cent. |
| Acute serofibrinous pericarditis | 14 | 7 | 50.0 | 4 | 28.6 | 3 | 21.4 |
| Acute purulent pericarditis..... | 44 | 21 | 47.7 | 9 | 20.4 | 14 | 31.8 |
| Subacute fibrinoplastic and purulent pericarditis..... | 14 | 5 | 35.7 | 5 | 35.7 | 4 | 28.6 |
| Total..... | 72 | 33 | 45.8 | 18 | 25.0 | 21 | 29.1 |

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The importance of empyema as an associated complication in the pericarditis of pneumonia is shown in Table 3.

TABLE 3.—EMPYEMA AND PERICARDITIS IN PNEUMONIA

| | | Empyema | | | | | |
|---|---|---|---|---|---|---|---|
| | | Bilateral | | Right | | Left | |
| | No. | No. | Per Cent. | No. | Per Cent. | No. | Per Cent. |
| Acute serofibrinous pericarditis | 14 | 4 | 28.6 | 5 | 35.7 | 1 | 7.1 |
| Acute purulent pericarditis | 44 | 8 | 18.2 | 9 | 20.4 | 24 | 54.5 |
| Subacute fibrinoplastic and purulent pericarditis | 14 | 5 | 35.7 | 3 | 21.4 | 4 | 28.6 |
| Total | 72 | 17 | 23.6 | 17 | 23.6 | 29 | 40.3 |

## TYPES OF INFECTION

A large proportion of the types of the pneumonia encountered were due to mixed infection by pneumococcus and streptococcus (usually one of the hemolytic varieties). When complications occurred in pneumonia, the predominating infection encountered was usually a hemolytic streptococcus; that is, 72.5 per cent. of 251 empyemas, in which the infection was known, were due to the streptococcus, while 27.5 per cent. were due to the pneumococcus. Such was the relative proportion also in pericarditis. In 75 per cent. of the cases, the cultures revealed hemolytic streptococci. In general, the type of infection corresponded to the type isolated from the pleural fluid.

## COMMENT

In Table 2 it will be noticed that in seventy-two instances of pericarditis, both lungs were involved in the preceding or accompanying pneumonia in 45.8 per cent., while the left lung was involved in 29.1 per cent. and the right lung in 25 per cent. McPhedran[1] has mentioned thirty-one cases of pericarditis from Osler's service at the Johns Hopkins Hospital, and has stated that both lungs had been affected in 41.9 per cent., the right lung in 41.9 per cent., and the left lung in 16.1

1. McPhedran, Alexander: Diseases of the Pericardium, in Osler: Modern Medicine, Philadelphia, Lea & Febiger 4: 41, 1908.

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per cent. From these figures, and from the experience of himself and others, he concluded that pericarditis occurred more frequently in association with affections of the right lung than of the left. This has not proved to be the case in the series here reported, which showed an almost equal proportion for the two lungs.

The most important factor in the development of pericarditis appeared to be the occurrence of empyema. Reference to Table 3 discloses the fact that pericarditis, especially the acute purulent form, occurred more frequently with empyema of the left pleural cavity (54.5 per cent. with left empyema, and 20.4 per cent. with right empyema). For all three forms of pericarditis described, it will be seen that this complication occurred with left empyema in 40.3 per cent., and with right empyema in 23.6 per cent. Pericarditis occurred more frequently in association with left empyema than with bilateral empyema. This may be explained by the fact that the development of empyema on the right side occurred, in a number of instances, as a late complication subsequent to the empyema on the left side. Pericarditis was one of the principal causes of death in empyema. Among forty-eight empyema patients who died subsequent to operation, extensive pericarditis, usually of the purulent form, was present in fifteen, or 31.2 per cent., while among 100 empyema patients who died without operation, pericarditis, usually of the purulent form, was present in 46 per cent.

### DIAGNOSIS

In the diagnosis of pericarditis, the clinical sign of greatest importance was a to and fro friction sound, not transmitted beyond a limited area, which was usually first heard over the base of the heart or in the fourth or fifth interspace close to the sternum, and which was practically synchronous with the heart beat. It possessed a distinct rubbing quality, not unlike but less perceptible than a pleural friction sound. The

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fremitus of the rub could in some cases be detected by palpation. Its duration was transitory if effusion occurred rapidly, when the heart tones became distant or muffled, and obliteration of the cardiac impulse occurred. The differentiation of the pericardial friction sound from a pleural or pleuropericardial friction rub, in a patient with dyspnea due to pneumonia, empyema or a partially collapsed lung, was a matter of great difficulty in numerous instances. In some patients, the friction rub could best be heard below the angle of the left scapula, because of the transmission through a portion of consolidated lung. The heart was in some instances distinctly rotated on its long axis toward the posterior chest wall, because of mediastinal adhesions arising from earlier mediastinitis. With effusion into the pericardium, the area of heart dulness increased to the right beyond the sternum, and to the anterior axillary line on the left. With marked effusion reaching 500 to 800 c.c., the area of dulness occupied a large portion of the left anterolateral chest wall. This area of dulness was often confusing if associated with the presence of fluid in the left pleural cavity. The roentgenogram was of great assistance in confirming the diagnosis of pericardial effusion in patients with increased area of heart dulness and with muffled heart tones, who also had walled-off fluid in the left lateral pleural cavity.

With marked distention of the pericardium, the veins of the neck distended, and in some instances a positive venous pulse in these vessels was evident, owing to the overfilled right auricle. At necropsy, dilatation of the right side of the heart was frequently mentioned in the necropsy notes. The myocardial changes, viewed microscopically, consisted of cloudy swelling and fatty degeneration, with fragmentation of the muscle bundles.

Since pericarditis occurred, in our experience, only one-eighth as frequently in patients without empyema

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as in patients with empyema, the occurrence of a possible purulent pericarditis was suspected in every empyema patient who presented, after pleural aspiration or drainage operation, a rapid pulse and a septic type of temperature. Dyspnea was so common a symptom in patients with an extensive pathologic condition of the chest associated with empyema that, by itself, little reliance could be placed on it.

In the diagnosis of small amounts of purulent material in the pericardium, the clinical signs were in numerous instances untrustworthy, and the roentgenograms of little assistance. It was not at all uncommon to see at necropsy the normal amount of pericardial fluid replaced with thin fibrinopurulent fluid. If the process had existed a number of days, as judged by the clinical history, fibrin organization had taken place in some instances, with firm villose adhesions between pericardium and epicardium, producing the pathologic picture of the "shaggy heart." There was usually a small amount of unorganized, thin pus in the pericardial cavity. This form of pericarditis was undoubtedly such from the beginning. In the presence of an empyema the condition appeared to have resulted from an extension by contiguity to the pericardium. In only two of the forty-four instances of purulent pericarditis did empyema not exist as an associated or antecedent condition, and in one of these a fibrinoplastic pleuritis was present, cultures from which revealed streptococcus infection.

The infection either reached the pericardium through the lymphatic vessels draining the parietal pleura, or extension occurred by contiguity of tissue from purulent accumulations beneath the sternum, which we have described as subcostosternal pus pockets, or from walled-off pockets occupying the left anterolateral portion of the chest cavity. In many of the patients with purulent pericarditis, the area of heart dulness was not increased by percussion, nor did the roentgenogram

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show cardiac enlargement, since distention of the pericardium had not taken place. There had merely been a change from a serous to a purulent fluid in the sac, with a subsequent partial organization of the exudate. The distant or muffled heart tones characteristic of considerably increased fluid in the pericardium could not be relied on for the diagnosis of the condition in such instances. A left-sided, closed pneumothorax, with collapse of the lung and retraction of the heart by adhesions toward the posterior chest wall, so muffled the heart tones in some patients as to lead to suspicion of pericardial effusion, when such a condition did not exist. A hemorrhagic pericardial fluid was obtained on aspiration in but one patient, who had undoubted tuberculosis with tuberculous pericarditis.

### TREATMENT

Since a large proportion of the deaths in this series occurred in patients with an extensive pathologic condition of the chest due to empyema, it was impossible to consider any more radical procedure than paracentesis in dealing with pericardial effusion, whether serous or purulent. In the acute, purulent form, as well as in the subacute fibrinoplastic and purulent form in which, in many instances, distention of the pericardium had not taken place, little could be expected from paracentesis or, for that matter, from free incision and drainage, because of the presence of the associated pulmonary and pleural pathology.

Pericarditis occurred as an early and as a late complication in pneumonia. As an early complication it was found at necropsy in patients who so quickly succumbed to a rapidly spreading pneumonia with empyema that the presence of pericarditis was unsuspected. As a late complication it occurred subsequent to operation for the relief of empyema. In not one instance among 275 operations for empyema was purulent pericarditis recognized, or believed to be coexistent with

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the empyema at the time of operation. It was many times suspected, but on evidence insufficient in the presence of pleural sepsis to warrant radical exploration of the pericardium. Subsequent events proved the wisdom of conservatism in the method adopted. In a strict sense, pericarditis developed, as did nephritis, subsequent to empyema and, as such, contributed largely as a cause of death in empyema, in many instances weeks after pleural drainage was established. If one might be so fortunate as to recognize purulent pericarditis coexistent with or subsequent to empyema, and the condition of the patient was such as to warrant further interference, free incision and drainage of the pericardium would be indicated. It has not fallen to my lot to be placed in such a position.

In performing paracentesis pericardii, local anesthesia of the skin and the deeper tissues was employed. Two points of election were available: A point outside the nipple, in the left fourth interspace, was the first selected. If the needle was inserted in an inward and backward direction toward the spine, keeping within the area of dulness and assisted by the roentgenogram shadow, there appeared to be little danger of puncturing the myocardium of the right ventricle, which occupied the greater portion of the heart surface proximal to the chest wall. The other point selected was immediately beneath the costal margin, in the narrow angle formed by it and the xiphoid cartilage on the left side. This point was available only if the diaphragm on the left side was in normal position. In many patients with empyema, the diaphragm was higher than normal on the affected side. In inserting the needle for aspiration at this point, it should be inclined so as to hug the under surface of the costal cartilage, and be pointed slightly to the left toward the nipple until it enters the pericardium.

A fairly good sized needle for attachment to the aspirating syringe should be selected. For persons

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with thick chest walls, the needle should be about 3½ inches long. When the point of the needle touched the myocardium, a distinct thrust, due to the cardiac systole, was evident.

In the treatment of pericarditis, the ice bag and morphin were used to control pain. Antistreptococcus serum, injected intravenously in from 50 to 100 c.c. daily doses, was used to combat the general sepsis in patients with streptococcus infection. Serums known to agglutinate, in dilutions of from 1:60 to 1:80, the strains of streptococci encountered, were used so far as possible. It is possible that recovery in some instances of empyema sepsis, with which suspected pericarditis was associated, was secured through its use.

## SUMMARY

1. Pericarditis was found to have occurred in seventy-two, or 24 per cent., of 300 patients who succumbed to pneumonia.

2. Acute purulent pericarditis occurred in 61.1 per cent. of the seventy-two instances, acute serofibrinous pericarditis in 19.4 per cent., while the subacute fibrinoplastic and purulent form producing the "shaggy heart" occurred in 19.4 per cent.

3. The quantity of pericardial fluid present in acute serofibrinous pericarditis varied from 250 to 1,000 c.c. With this form, pneumonia of both lungs was present in 50 per cent. of the patients, of the right lung alone in 28.6 per cent., and of the left lung alone in 21.4 per cent.

4. In acute purulent pericarditis, the quantity of pus in the pericardium varied from 100 to 1,000 c.c. The average in twenty-six instances in which the amount was recorded was 350 c.c. Pneumonia involving both lungs had occurred in 47.7 per cent. of the cases, involving the left lung alone in 31.8 per cent., while in 20.4 per cent. the right lung was alone

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Fig. 1.—Pericarditis following pneumonia. Three aspirations of serofibrinous fluid. Recovery.

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Fig. 2.—Hemorrhagic pericardial effusion following tuberculous pneumonia.

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Fig. 3.—Pericardial effusion and left encapsulated empyema. Operative Series II, No. 3. Recovery.

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Fig. 4.—Purulent pericarditis with rotation of heart toward posterior chest wall, due to adhesive mediastinitis. Five aspirations. Recovery, but with marked disability.

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involved. In 54.5 per cent. of these instances, empyema of the left pleural cavity was present.

5. In the subacute form of fibrinoplastic and purulent pericarditis, producing the so-called "shaggy heart," pneumonia of both lungs had occurred in 35.7 per cent. of the cases, of the right lung in 35.7 per cent., and of the left lung in 28.6 per cent. Bilateral empyema had occurred in 35.7 per cent. of these instances, right empyema in 21.4 per cent., and left empyema in 28.6 per cent.

6. The type of infection isolated from the pericardial fluid corresponded generally to the type of infection isolated from the pleural fluid. In about 75 per cent. of the patients streptococcus infection (usually one of the hemolytic varieties) was present.

7. The most important factor in the development of pericarditis appeared to be the occurrence of empyema. Pericarditis appeared to have resulted by contiguous extension from an empyema in many instances. Pericarditis and the associated myocardial changes were important contributory causes of death in empyema, both prior to and subsequent to operation.

8. In the diagnosis of pericardial effusion in amounts varying from 300 to 500 c.c., the physical signs and the roentgenograms were fairly dependable, despite the extensive, associated pathologic condition which confused the picture. In the diagnosis of smaller amounts (from 10 to 150 c.c.) of purulent material in the pericardium, the clinical signs were untrustworthy and the roentgenograms of little assistance.

9. Paracentesis of the pericardium was an important diagnostic and prognostic procedure. In acute serofibrinous pericarditis with large effusion, aspiration of the fluid was an important means of securing relief for the cardiac embarrassment.

10. The more radical procedure of incision and drainage of the pericardium in purulent pericarditis

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was not considered advisable in any of these patients, in which the condition was suspected, because of the extensive, associated pathologic condition.

217 Nasby Building.

## ABSTRACT OF DISCUSSION

DR. CHARLES SPENCER WILLIAMSON, Chicago: Just before the entrance of the United States into the war I was engaged in the experimental study of pericarditis and to this end injected gelatin agar mixtures into the pericardial cavity of thirty-three fresh cadavers, and then made casts from them. These casts were exhibited at the New York meeting of the American Medical Association in the scientific exhibit. To one of these cases I wish to refer since it was a unique opportunity. A patient with an upper lobe pneumonia developed a pericardial rub and shortly afterward we made the diagnosis of fluid being present. The following day, hearing that the patient was rapidly growing worse, I went to the hospital and found him with a very rapid, weak pulse and in a dying condition. Before we could make plans to aspirate the patient died. A few minutes after death I made a small incision in the costoxiphoid angle, inserted a long needle and aspirated 270 c.c. of turbid fluid containing pneumococci. I immediately injected exactly the same amount of gelatin agar solution and twenty-four hours later removed the pericardium en masse. This was frozen and a cast and model made. The size of the exudate as measured on the model was 405 c.c. The important point is that even with this small exudate the great veins and the auricles were absolutely flattened. Practically we had a repetition of the animal experiments of Francois, Franck and Starling. The fluid had accumulated so rapidly that the pericardium did not distend sufficiently and in my judgment the patient died from the mechanical interference of the circulation due to pressure on the auricles. The important therapeutic indication is to remember that when an effusoin is poured out rapidly the condition of the patient may suddenly become alarming and aspiration of even a very small quantity of fluid may be lifesaving. In other words, a small effusion accumulating rapidly may kill the patient where a large effusion accumulating slowly may be well borne.

DR. C. F. HOOVER, Cleveland: Paracentesis may be done for relief of infection of the pericardium or for relief from pressure within the pericardial sac. Whenever there is a question of performing paracentesis for the relief of pressure within the pericardial sac I am guided by the movements of the costal borders from the subcostal angle to the eighth rib. When the subcardial part of the diaphragm is flattened there will be inspiratory narrowing of the subcostal angle.

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If the subcardial portion of the diaphragm is not flattened then there will be no need of relieving the pericardial pressure. Another sign I have found very valuable in estimating the need of paracentesis of the pericardial sac is pallor of the lips. When the circulation is failing on account of pressure in the sac the lips are pale and the pulse small. Pallor of the lips is not caused by failing circulation when the myocardium is at fault. Distension of the pericardial sac causes greater hepatic displacement than any other intrathoracic disease. The liver is rotated downward on a transverse axis which is located at that part of the liver not covered with peritoneum. Seven hundred cubic centimeters of fluid in the sac was sufficient on one occasion to rotate the liver downward until the lower border was in the iliac fossa.

Dr. William H. Robey, Jr., Boston: I have been very much interested in the remarks of the speakers, especially in what Dr. Hoover said, because I think that any light which can be thrown on the diagnosis of fibrinous, serous or purulent pericarditis is of great value. I have noted in the plates which Dr. Stone showed that the cardiohepatic angle was not obtuse. It is more often obtuse in large serous effusions than in small purulent ones. I do not think it is the opinion of observers that relatively large amounts of fluid in the pericardial sac produce signs of pressure. It is only when the fluid is first forming before the distensible pericardial sac has accommodated itself to the increased pressure or when the amount is very large that there are signs of discomfort. At the meeting last year Dr. Christian reported some interesting cases of posterior accumulations of fluid which caused signs of compression of the left lung within the angle of the scapula, known as "Ewing's sign." I recently saw in France a case with thirty-two ounces of pus in the sac and because the cardiac impulse was perfectly distinct the surgeons would not believe that the pus came from the pericardial sac even after operation. In another case with pericardial rub followed by increasing cardiac area, Ewing's sign was present. There was a dry tap and at the necropsy the pericardial surfaces were adherent. Ewing's sign failed in this instance. I notice that Dr. Stone and some others caution physicians not to puncture the heart wall in aspiration. I have often gone into the heart wall and have never seen it do any harm. In looking through the literature I have found only one case where the patient bled to death because the needle entered the ventricle. Indeed, in small amounts of fluid, the pericardial space being purely potential it is almost impossible not to strike the heart wall. One of my friends told me that he has given immediate relief to cases of marked cyanosis and dyspnea by aspirating the embarrassed heart and has never seen anything but good come from the operation.

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Dr. Henry Sewall, Denver: A pericardial effusion may affect the welfare of the individual in two ways, namely, through the quality of the fluid, its infectiveness, or through its amount. A noninfective effusion works its harm by preventing full diastolic distension of the heart. The restraint thus offered depends on the relation of two factors, namely, volume of the fluid and capacity of the pericardial sac. The latter factor is itself variable but data on the conditions of pericardial distensibility seem to be wanting. It is conceivable that a pericardial effusion of moderate amount may cause sudden death by pressure on the great veins entering the right auricle while the patient lies on his back. In such a case the obstructive pressure might presumably be relieved by turning the patient on his face. Some of the speakers are to be congratulated on their immunity from harm in puncturing the heart itself during paracentesis. It seems to be a question of luck. I recall a case of cardiovascular disease in which the left pleural cavity was tapped by a well known surgeon for removal of serous exudate. The patient died in a few hours; the necropsy disclosed a pericardial sac distended with blood. The aspirating needle had cut into a superficial coronary vessel on the left ventricle and the patient bled to death into her pericardium. We all remember Kronecker's demonstration of the localized coordinating center in the middle of the anterior surface of the right ventricle, irritation of which, in the dog, produced fibrillation of the heart.

Dr. Willard J. Stone, Toledo, Ohio: I have never seen displacement of the liver downward in extensive pericardial effusions because practically all of our cases, at least seven-eighths of them, were associated with empyema in which high fixation of the diaphragm had occurred on the affected side. Such high fixation would operate to prevent downward displacement of the liver if associated with rightsided empyema. In connection with the description which Dr. Robey mentioned of Dr. Christian's paper last year before this section, one of the plates this morning showed a distinct rotation of the heart toward the posterior chest wall. In that case the signs of effusion and dulness were very marked in the back. We have believed in such cases that an adhesive mediastinopericarditis had occurred which had caused rotation of the heart toward the posterior chest wall accounting for the physical signs which were found in the back. These signs were accentuated if transmitted through a portion of contiguous consolidated lung. I do not believe that the sign described by Ewing is of any great significance in connection with pericarditis, except as it explains the associated chest pathology. I am sure, in doing paracentesis of the pericardium, that I have punctured the ventricle of the heart a number of times. I do not know that any particular harm

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has followed the incident, but believe, of course, that it is just as well to avoid puncturing the ventricle if it is possible to do so. To carry a needle through a thickened septic pericardial membrane, through the ventricle and into the blood stream is hardly a very desirable surgical procedure. If one uses the roentgenogram as a guide and keeps within the area of heart dulness, a point to the left, below and just outside the nipple, is on the whole the most satisfactory point for aspiration. The angle between the xiphoid and the left costal border was rarely available in our series because of the high diaphragm associated with empyema. I should like to ask Dr. Robey if he has ever seen any harm come from puncturing the ventricle in pericardial paracentesis.

Dr. William H. Robey, Jr.: Not in my experience or in that of my friends, and I have found but one case in the literature where any harm seemed to have come from it.

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# STUDIES ON MALARIA CONTROL

## IX. EFFECTIVE AND PRACTICAL TREATMENT OF MALARIA TO DISINFECT INFECTED PERSONS AND TO PREVENT RELAPSE *

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NEW ORLEANS

When a physician is called on to treat a case of malaria, usually the chief object he has in view is to relieve all clinical symptoms. The disinfection of the patient and the prevention of relapse receive secondary consideration, if any. There is great variation in the treatment of clinical cases of malaria, and still greater variation in the treatment for the purpose of disinfecting the patient. Disinfection is of greater importance than it is generally considered to be, because of the fact that if not disinfected the patient is likely to have latent malaria or to relapse with more or less harmful effect, and because of the further fact that during all the time he is a potential source of infection to others.

In a study of the frequency of malaria relapse made on 3,815 persons in Sunflower County, Mississippi, during 1918, the information obtained showed that between 50.77 and 68.86 per cent. of all persons who had attacks of malaria during the year had relapses and not new infections. It is not uncommon for persons to have from one to several attacks of malaria every year, for a period of two or three years. There are plenty of instances in which the possibility of reinfection was entirely eliminated,

* From the Department of Experimental Medicine, Tulane Medical College.

*This is one of a series of papers to be published, based largely or entirely on malaria control work conducted jointly by the International Health Board and the Mississippi State Board of Health.

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showing that they were relapses and not new infections.

The importance of disinfecting infected persons has not received as much consideration by practitioners as it deserves. Using the data that have been obtained in a study of malaria control in the Mississippi delta during the past three years, I have been able to estimate with a fair degree of accuracy the reduction in the prevalence of malaria that would take place if all physicians disinfected all the cases they treat. It was shown that malaria would be reduced approximately 90 per cent. in ten years' time by this means alone.[1] Every case of malaria means that the patient will continue as a potential source of infection until disinfected, and every malaria patient disinfected means the destruction of one or more sources of infection.

Malaria is one of the diseases in which the clinical symptoms are relieved a long time in advance of the disappearance of the specific infectious agent. No high degree of effective immunity is conferred by an attack of the disease in the majority of instances. Whenever sufficient treatment is given to reduce the number of parasites sufficiently, the symptoms cease, but the infection continues. Small numbers of parasites may remain in favorable locations in the tissues of the body, and probably continue to reproduce. Later, when conditions become more favorable, they may, and often do, give rise to a return of clinical symptoms, or relapse.

Though blood examination demonstrates the presence of parasites in practically all cases of clinical malaria, and in many instances of persons who are infected but have no recognized clinical symptoms at the time, it fails to demonstrate their presence in many persons who have smaller numbers but are still infected. The number of parasites may be so small

1. Bass, C. C.: Studies on Malaria Control, V, The Importance of Disinfecting All Cases Treated as a Factor in Malaria Control in a Locality of Great Prevalence, South. M. J., to be published.

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that no practical examination of the blood would demonstrate their presence. As long as the patient has one viable parasite in him, he remains infected. Treatment for the purpose of disinfection is not effective, therefore, until every parasite is destroyed. When this is accomplished, no relapse occurs, nor is the patient a source of infection to others. Treatment which will destroy every parasite, and which is practical at the same time, is my subject here.

The disappearance of clinical symptoms and of parasites from the peripheral circulation cannot be depended on, therefore, to show when disinfection has been accomplished. In many instances, patients carry the infection for months, sometimes as long as a year, or even longer, without showing any clinical symptoms whatever. Then something happens that reduces the effectiveness of the means by which sufficient resistance has been maintained to retard the multiplication of parasites, and clinical symptoms of malaria develop. Such things, for instance, as sudden chilling of the surface of the body, getting wet, eating large quantities of indigestible food, the shock of surgical operations, or injuries, are frequently sufficient to precipitate attacks in persons who are infected but who have been able to resist the disease sufficiently to prevent clinical symptoms.

### THE DURATION OF PROPER QUININ TREATMENT

The duration of proper quinin treatment is the only reliable guide as to when disinfection has been accomplished. Since the disappearance of clinical symptoms and of parasites in the peripheral circulation, presence of which affords the basis for a diagnosis of malaria, cannot be depended on as a guide as to when disinfection has been accomplished, we must rely on methods of treatment which have been demonstrated by experience to prevent the occurrence of relapse. Quinin is the only drug known which destroys malaria parasites

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and is capable of disinfecting. It is, fortunately, a very perfect specific, and I believe it can confidently be said that when properly used it will disinfect any patient. My opinion is that there are no exceptions. Perhaps the amount of quinin and the duration of administration required may, in exceptional cases, be much greater than what the average patient requires; but there surely is a dose and a duration that would be effective in every instance. Failure simply means that proper doses of quinin have not been administered or that there has not been proper duration of treatment, and not that the patient cannot be disinfected by treatment with quinin.

### AVOIDANCE OF INCONVENIENCE AND DISCOMFORT

Practical treatment must be convenient and must not produce great discomfort. Even after the acute symptoms of malaria have been relieved, the patient must be given quinin for a considerable length of time to make reasonably certain that he is disinfected. On account of the fact that the treatment must be so long continued, long, in fact, after the patient thinks he is well, the quinin must be given in a convenient form. During the prolonged treatment the patient is usually up and about at his usual occupation, and any treatment that puts him to considerable inconvenience would be much less likely to be continued than one that did not. Quinin is a drug that is slowly absorbed and slowly eliminated, and it is not necessary, therefore, that it be given at frequent intervals. For the purpose of disinfection, one dose a day is just as effective as any larger number of doses, and it is much more convenient to take one dose daily than a larger number. Generally speaking, it is much more convenient to take it at night before retiring than at any other time of day.

If the treatment produces great discomfort it is also not as likely to be carried out as if it did not. When

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given hypodermically, great discomfort is produced and, in practice, few patients are ever disinfected when this method of administration is prescribed. Sometimes quinin is given by mouth in acid solution. This is one of the most nauseating doses, and any physician who has been in the habit of prescribing quinin in this form, or who contemplates doing so in the future, should take it for a while himself. He will then be better able to decide whether or not to prescribe its use in this fashion. It is much better to give it in the form of tablets, for those who can swallow tablets, and in the more or less tasteless form of quinin in aromatic syrup of yerba santa for young children. The discomfort caused by quinin injection and by taking it in acid solution by the mouth is often a cause of abandoning the treatment before the patient is disinfected.

Quinin causes considerable discomfort, such as tinnitus, urticaria and nerve irritation, in a great many persons. Susceptibility to these unpleasant effects varies greatly with different persons. Some are rendered quite uncomfortable by even small doses, while others may take considerably larger doses without discomfort. Fortunately, there is, in the great majority of instances, no great unpleasantness produced by the amount necessary for disinfection. It is very important that the necessary dose should not be greatly exceeded because of the resulting discomfort. Otherwise, the treatment is much more likely to be abandoned before the desired results have been accomplished. There is no other specific remedy for malaria and, therefore, no other treatment to choose. Those who are affected unpleasantly by it are unfortunate, but still their only specific remedy is quinin. The sooner this fact is learned and appreciated by the medical profession, as well as the laity, the better it will be for malaria sufferers.

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SPECTACULAR METHODS AND UNUSUAL SALTS

Since the treatment must be kept up for a considerable length of time to disinfect, and since the success depends very much on the patient's confidence and cooperation, spectacular methods and special salts and preparations of quinin should not be used, even if they should be as effective, dose for dose, which they often are not. So far as I have been able to learn from experimental studies, and from observation of treatment of those who have malaria, in a group of more than 40,000 persons living in a heavily infected section, there is no salt of quinin any more effective in disinfecting a patient than ordinary sulphate, and there is no method of administration that is as effective or practical for this purpose as administration by mouth.

During 1916 and 1917, those who were found to have malaria among 31,459 persons examined in Bolivar County, Mississippi, were furnished quinin in different forms and in different size doses, to be taken for variable lengths of time in different groups. A blood examination was made of these persons subsequently, to determine the results of the different methods of treatment. From the data obtained, it appears that 10 grains of quinin a day for a period of four weeks disinfects about 60 per cent., and that 10 grains a day for eight weeks disinfects about 90 per cent. There were some instances of relapse in the case of persons who took the full eight weeks treatment faithfully.

It was also found that patients take 10 grains of quinin daily better, and more systematically, than they will take larger doses, such, for instance, as 20 grains daily. They will not take the large dose so well because of the discomfort produced, including, especially, tinnitus and dizziness, which often occur in laborers and in people under the influence of quinin working out in the heat of the sun.

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As a result of this experience, we have adopted 10 grains of quinin daily, to be taken, preferably, before retiring, for a period of eight weeks, as the minimum dose that can be depended on to disinfect. (Children should be given a proper proportionate dose.) Patients thought likely to be especially resistant should be given a somewhat longer course of treatment. If judgment and care are used, most of the patients who would fall in this group can be found by giving attention to the history, which usually tells of previous relapses. Patients who give a history of previous relapses should be put on longer than eight weeks treatment.

## SUMMARY

Disinfection of the patient has not received the consideration that it deserves in the treatment of malaria.

The disappearance of clinical symptoms and of demonstrable parasites in the blood cannot serve as a reliable guide as to when disinfection has been accomplished. Nor can any other known method of examination determine when the infection is eliminated. The most dependable guide at our command is the duration of a treatment that experience has shown to be effective.

Quinin, in sufficient doses for a sufficient period of time, is effective in all cases. I believe that there are no exceptions to this statement. Instances in which the treatment that is successful in average cases fails to disinfect are examples, not of the failure of quinin to disinfect, but simply of the failure of quinin in that particular dose and for that particular length of time. If the treatment be continued for a sufficient length of time, it always succeeds, even in these exceptional cases.

The treatment, to be practical, and to be carried out by the patient, must not cause great discomfort or inconvenience. One dose at night is usually more con-

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venient than a larger number of smaller doses, and it is equally effective. Less discomfort is produced also.

The treatment recommended, one to disinfect infected persons and to prevent relapse, is 10 grains of quinin sulphate at night, before retiring, for a period of eight weeks. When there is good reason to think a given patient will be more difficult to disinfect than the average one, the treatment should be continued considerably longer than eight weeks.

NOTE.—What has been said refers to the treatment of cases without acute symptoms, or after active symptoms have been relieved. Active clinical symptoms are relieved in practically all cases by three doses of 10 grains of quinin, during each day, for three or four days.

## ABSTRACT OF DISCUSSION

DR. C. S. WILLIAMSON, Chicago: We do not see very much malaria in Chicago, and yet when we do see it, it presents some interesting problems. During the past five years three cases have come under my care under almost identical conditions. The patients were young men; the diseases came on after exposure to cold in February, and the diagnosis in each case was lobar pneumonia. None of these patients had been out of Chicago for many months. None of them knew that they had had malaria before; the diagnosis was confirmed by blood examination. The symptoms were such as to lead inevitably to a diagnosis of lobar pneumonia. In one case the patient was given quinin sulphate for five months in doses of about 30 grains a day, followed by an acid solution, and at the end of that time there was a relapse. The patient had not left Chicago.

Will Dr. Bass tell us whether he believes that the temperature of Chicago might reawaken an old malarial infection or whether there is some particular strain of the parasite which must be accounted for. These cases impressed themselves on my mind because it seemed to be almost impossible to have malaria in the cold winter. One patient was working on a bridge, and you would naturally think of pneumonia and not of malaria and mosquitoes.

DR. CHARLES C. BASS, New Orleans: I do not think that we know just how exposure to cold and other influences that lower the vitality of the individual will cause relapses. It is a most common experience where there is much malaria that, given a group of men many of whom are infected and who are getting along well, if it rains and many of them get wet, sudden chilling of the surface of the body is followed in three or four days by chills and fever, although

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the patients had not had chills and fever before; but this sudden chilling of the body seems to affect them. Another observation that most of us who have studied malaria have made is that a constant even temperature, when there is little variation between the noon to night temperature, not as many malaria relapses are noted as during the early season of the year and later in the fall when there is great variation in the extremes of temperature. It seems reasonable to suppose that this is one of the many things that may lower the general vitality of the individual and in some way interfere with the resistance by which he holds back or prevents relapse. The shock of a surgical operation may do the same thing. Perhaps, however, when we know more about the immunity processes by which individuals are able to prevent relapses and eliminate the relapses without treatment the relapses will be fewer.

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# THE DIAGNOSIS OF CHRONIC PULMONARY TUBERCULOSIS*

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The recognition of chronic pulmonary tuberculosis is generally regarded as a simple matter in which there is slight chance of error. That this is not always the case is quickly recognized by any one who has the opportunity of studying a series of cases of advanced pulmonary disease. This study is based on our experience in the department for diseases of the chest of the Jefferson Hospital. In this department the dispensary handles all stages of tuberculosis, early and late, but only advanced cases are admitted to the wards. By admitting only these it seemed that we were making the best use of the beds in the effort to lessen infection by isolating patients in the advanced stages. Our admissions come from the general medical clinic and from the tuberculosis dispensary, but a considerable number are accepted on the diagnosis of other hospitals and general practitioners. We have no opportunity to study these cases until after admission. This study is made to check the correctness of the diagnosis of advanced pulmonary tuberculosis as made by many different members of the profession.

It may be asked what difference an error makes if the patient has some chronic disease. In some of the conditions, for example, bronchiectasis, it is evident that no great harm will be done; but, in others, valu-

* From the Jefferson Medical College.

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able time may be lost and a condition left untreated which might be helped. Syphilis and the presence of a foreign body in the bronchus are examples of such a condition. In addition, such patients are occupying beds which might be more usefully employed. In discussing errors in diagnosis, we must realize that every one makes mistakes, and we should be charitable to the other man concerning those he makes, but very severe in judgment on our own. The important lesson is to try and reduce the number of errors in the future.

This report is based on the study of 1,200 consecutive admissions, all the patients coming with a diagnosis of advanced pulmonary tuberculosis. In the series, seventy-two, or 6 per cent., were found to be nontuberculous. There is always a chance of error in a clinical diagnosis, but we have a check in the necropsy results. There were 134 necropsies in this series, and among these it was found that seven patients had been nontuberculous, 5.2 per cent., which is fairly close to the previous figure. Of these seven cases which came to necropsy and were found to be nontuberculous, in five we had made the correct diagnosis; but two went to necropsy with a diagnosis of tuberculosis in which we were in error. These cases are discussed later.

There is an interesting study by Ash[1] on the necropsy findings at the Boston Consumptives' Hospital. In 198 necropsies, twenty-three cases (11.6 per cent.) were found to be nontuberculous; seven of these had been correctly diagnosed before death. He collected a series of 353 necropsy cases from eight hospitals and found that thirty-eight (10.9 per cent.) of these were nontuberculous. The total of this report gives 551 necropsies with sixty-one nontuberculous cases (11 per cent.). The conditions found were: chronic cardiorenal, 27; pneumonic sequelae, 11; neoplasm, 7; aneur-

1. Ash, J. E.: The Pathology of the Mistaken Diagnoses in a Hospital for Advanced Tuberculosis, J. A. M. A. 64: 11 (Jan. 2) 1915.

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ysm, 6; syphilis, 4; septicemia, 4; actinomycosis, 1; pellagra, 1.

In our series, the various conditions which were wrongly diagnosed as advanced pulmonary tuberculosis are: cardiorenal, 19; pneumonic sequelae, 9; bronchiectasis, 8; abscess of lung, 8; chronic bronchitis, 6; neoplasm, 5; syphilis, 4; aneurysm, 2; anthracosis, 2; bronchial asthma, 2; empyema, 2; diabetes mellitus, 1; cancer of rectum, 1; foreign body, 1; malingering, 1.

Seven of these came to necropsy (five with a correct diagnosis), the conditions found being: abscess, two cases; cardiorenal disease, two cases, and one each of aneurysm, neoplasm, and delayed resolution in lobar pneumonia. Of the two instances in which cases came to necropsy with a diagnosis of tuberculosis, one of the patients had had chronic lung abscesses, and the other, delayed resolution of a lobar pneumonia.

It is to be understood that there were cases in which these various diseases, with the exception of syphilis and neoplasm, were associated with tuberculosis. We have given only the number of cases in which an error in diagnosis had been made and tuberculosis was not present. The various groups are discussed in detail.

### VARIOUS GROUPS

1. *Cardiac and Cardiorenal Disease* (nineteen cases).—These are cases in which a proper diagnosis might give a chance of benefit, in some cases, at any rate. The reasons for error are very evident, and first among them we must put careless or incomplete examination. These are the cases in which there is the least excuse for error. Failing health and strength, dyspnea, cough and the finding of râles in the chest were the features which were wrongly interpreted. A proper physical examination and a study of the sputum should prevent such mistakes. This group took first place in the series of instances collected by Ash. Of

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the cardiac cases it is of interest to note that fifteen were cases of mitral disease, in seven, mitral stenosis being present.

2. *Chronic Inflammatory Conditions in the Lungs, Usually Associated with Some Form of Pneumonic Process* (nine cases).—There was usually an associated general bronchitis. It is evident that in this group there are difficult problems, but it may be that because of this difficulty more care is taken. Of these cases, three proved to be unresolved lobar pneumonia, and six were atypical cases of bronchopneumonia. Case 1 is an example of lobar pneumonia in which we failed to make a correct diagnosis:

CASE 1.—W. G., a negro, aged 37, was admitted with a history of cough which for three months had become progressively worse. Three weeks prior to admission he was compelled to go to bed because of chilliness, fever and weakness. His cough became quite severe and was associated with blood-tinged sputum. His best weight was 175 pounds a year before, and for four months he had progressively lost weight until, on admission, he weighed 99 pounds. Examination showed an extremely sick, emaciated negro with signs of consolidation over the greater part of the right lung and scattered, crackling râles throughout both lungs. One sputum examination was negative. A clinical diagnosis of tuberculous pneumonia, in spite of the one negative sputum, was made, because it was thought that the finding of tubercle bacilli had perhaps been missed, but would show on future examinations. The patient died on the third day following admission.

The necropsy revealed the following nontuberculous changes: The right lung was solid except along the anterior margin and extreme apex of the upper lobe, anterior margin of the middle lobe, and a slight fringe at the bottom of the lower lobe. An incision of the organ showed surfaces that were gray, except a small portion of the apex and the base of the lower lobe; these areas were light red, with small, grayish mottlings. The gray areas were firm. The left lung was large, dark in color, and very soft, with decreased crepitation. The incised surface exuded an excess of frothy, blood-stained serum. The pathologic diagnosis (verified by histologic examination) was lobar pneumonia with gray hepatization of the right lung.

Several cases were instances of the slow-clearing basal pneumonia to which much attention has recently

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been drawn. The good rule that a basal process is rarely tuberculous is often forgotten. Case 2 is typical:

CASE 2.—E. S., a white woman, aged 24, referred with a diagnosis of advanced pulmonary tuberculosis, had lost about 25 pounds in the past year, and two weeks before she began to cough and spit blood. The temperature averaged from 102 to 103. There were signs of acute bronchitis, with many râles throughout both lungs. The resonance was impaired at both bases with suppressed breathing and numerous fine, crackling râles. Sputum examinations were repeatedly negative for tubercle bacilli. After one month her temperature became normal and remained so for another month, at the end of which time she had a febrile recurrence with the same symptoms and signs. The condition again cleared up in about four weeks, and the patient was discharged three months later free from symptoms and signs. During the first febrile period the following roentgen-ray findings were noted: There was a very dense area at the base of the right lung, probably unresolved pneumonia, and another extending from the second to the fourth rib anteriorly and the fifth and sixth posteriorly. The left base showed some clouding but not as extensive as on the right side. . The apexes appeared fairly clear.

With regard to the subacute and chronic nontuberculous pulmonary infections, the composite clinical history of a group of these patients is about as follows: The patient has a cold with slight fever but continues at work, although he feels ill, or, in more severe cases, may be compelled to go to bed for a few days or a week. A diagnosis of bronchitis or influenza is made. A week or ten days after the onset the fever is less, although there may be an afternoon elevation between 99 and 100. The constitutional symptoms subside, but cough and expectoration continue and, in a few instances, there may be hemoptysis. Examination reveals a more or less constant localization of signs in the lower lobe, dulness, increased vocal fremitus, diminished breath sounds and many fine, crackling râles. Tubular breathing is seldom heard. The sputum examination is negative for tubercle bacilli, but usually shows a larger number of other organisms.

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This group has been carefully studied, and the differential diagnosis rests, as Miller states, on the constant localization in a lower lobe, the absence of constitutional symptoms in the presence of extensive physical signs, the absence of tubercle bacilli, and the presence of the organisms of acute respiratory infections, the character of the roentgenogram, and the disappearance of signs and symptoms, in the majority of instances, far more quickly than would be possible in tuberculous lesions of similar extent.

It is of interest that most of our cases of this group came in during the past winter, and this is undoubtedly because of the influenza epidemic. Many errors of the nature described are to be made in the next few years if the experience of the last pandemic is any guide. In the nineties we learned by experience that there were many atypical pneumonias of long duration, sometimes apical, which were mistaken for tuberculosis. In many of these, the question of diagnosis is concerned with the possible lighting up of an old tuberculous process and, in this, the roentgen rays give little help in some cases. We would emphasize the value of time and repeated sputum examinations in making a correct diagnosis in this group.

3. *Bronchiectasis, Usually with Marked Fibrosis* (eight cases.)—It is easy to understand the reason for this error if the diagnosis is made on physical signs alone and the sputum is neglected. The study of the sputum should prevent mistakes. We have reported one of these cases[2] in which the patient had been in three tuberculosis sanatoriums with a diagnosis of advanced pulmonary tuberculosis.

4. *Pulmonary Abscess* (eight cases.)—Apparently the general features, fever, emaciation and sweating, with the signs in the lung, were relied on for diagnosis.

2. McCrae, Thomas, and Funk, E. H.: Bronchiectasis of the Upper Lobes, J. A. M. A. **67**: 1060 (Oct. 7) 1916.

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The examination of the sputum was evidently neglected. Sputum which is persistently purulent and does not contain tubercle bacilli is strong evidence against tuberculosis. In one case of this group we made an error in diagnosis:

CASE 3.—J. D., a white man, aged 32, gave a history of pleurisy three years before, since which time he had a persistent cough with expectoration. Ten months previously he had been in a tuberculosis sanatorium. He had a pulmonary hemorrhage one week prior to admission. He had lost about 16 pounds in weight. He had not had chills or sweats, but had been feverish in the afternoon for some months. The examination disclosed, in addition to the signs of acute bronchitis, evidence of infiltration and cavity formation in the left lower lobe. The sputum examination was reported positive for tubercle bacilli. In spite of the basal character of the lesion, which our clinical sense told us was against tuberculosis, we made a diagnosis of tuberculosis on the sputum examination. The necropsy revealed a large cavity in the lower left lobe, the outer wall of which was the greatly thickened pleura. The cavity had a fairly distinct wall with a dilated bronchus opening into it. The nonadherent portion of the pleura was covered by a grayish exudate. There was no evidence of tuberculosis in either lung.

5. *Emphysema and Chronic Bronchitis* (six cases).—Reliance on symptoms and signs, together with neglect of proper sputum examination, is the explanation of errors in this group.

6. *New Growth* (five cases.)—The same remarks apply as in the two preceding groups. Error is due to lack of proper examination, especially of the sputum. An example is presented in Case 4:

CASE 4.—A. T., a white man, aged 40, was referred with the diagnosis of advanced pulmonary tuberculosis. He gave a history of onset one year before with cough, expectoration, hemoptysis, marked gastric symptoms, and loss of weight. The physical examination revealed marked emaciation, with signs in the chest indicative of consolidation of the upper half of the left lung and scattered râles throughout the entire pulmonary area. The sputum examinations were repeatedly negative. The Wassermann test was negative. A careful study of the history revealed the fact that the gastric symptoms were primary and had been quite marked since the onset. There had been pain after eating, vomiting of brownish material, etc. Examination of the abdomen revealed a

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small mass in the region of the pylorus which the roentgen ray confirmed as a pyloric tumor. The sputum examinations continued negative, and a diagnosis of secondary carcinoma of the lung was made.

7. *Syphilis* (four cases.)—With the present craze, in some quarters, to diagnose syphilis of the lung on rather slight evidence, the number of cases under this heading may seem small; but we are conservative in making this diagnosis. These cases seemed thoroughly proved, and the results of specific treatment were striking. An example is given:

CASE 5.—L. H., a colored woman, aged 27, referred by a general hospital with the diagnosis of advanced pulmonary tuberculosis with laryngeal involvement, gave a history of cough which, for two years, had gradually grown worse, and of expectoration which at times was blood tinged. During the two weeks before admission she had many small hemoptyses. She was weak and had lost a good deal of weight. In the past year she had been treated in a New York hospital for advanced tuberculosis. The physical signs and roentgen ray showed fairly marked bilateral pulmonary disease. The temperature usually rose to about 101 in the afternoon. The sputum was repeatedly negative for tubercle bacilli, and the laryngologist reported that the laryngeal ulceration seemed more like that due to syphilis than tuberculosis. The Wassermann test showed a strongly positive reaction. Following antisyphilitic treatment, the symptoms disappeared, the temperature fell rapidly to normal, the signs became less and, four years later, she is well and working.

8. *Aneurysm, Anthracosis, Bronchial Asthma* and *Empyema* (two cases of each).—In both cases of aneurysm the diagnosis, while suggested by symptoms and signs, was made positively only by the roentgen-ray examination. Anthracosis is regarded as tuberculosis much more commonly than these figures indicate, and many such cases sent to the hospital as tuberculosis were recognized in the dispensary and sent to the general wards; so they do not figure here. With any care in sputum examinations, such an error should not be made. The cases of empyema were not the ordinary type, but were interlobar and, while difficulty in the diagnosis is understood, sputum examinations would have prevented the error.

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9. *Miscellaneous Cases* (one each of diabetes mellitus, cancer of the rectum, foreign body in a bronchus and malingering).—In the first two cases there was some cough, but little else in the way of signs even to suggest the diagnosis. The foreign body was a bullet in the bronchus, the diagnosis being made by the roentgen ray. In several other cases of foreign body which were sent for admission, suspicion was aroused and the patients were not admitted. This figure does not, therefore, give an accurate idea of the number of cases in which an erroneous diagnosis had been made. The malingerer was well up in the symptoms of the disease and was found to have scratched his throat to produce blood-streaked sputum. It was found that he had done this in other hospitals and had been regarded as tuberculous. His object was to evade the military draft.

COMMENT

What were the reasons for these errors? Lack of thorough examination, drawing hasty conclusions from symptoms and signs, and neglect of proper sputum examinations are probably the main causes. Many of these conditions result in loss of weight and strength with anemia, are accompanied by fever, and show signs in the lungs. The great safeguard is proper examination of the sputum. In any patient with marked signs in the lungs and a sputum negative for tubercle bacilli, suspicion of a diagnosis of pulmonary tuberculosis should be aroused at once. Too often a diagnosis is made and no thought given to proving it. How many negative sputum examinations are necessary before tuberculosis can be excluded? Six negative examinations are said to be enough, but we should suggest more than this.

The problem is very different from that concerned in the diagnosis of early pulmonary tuberculosis. Here we should endeavor to make the diagnosis before

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bacilli are found in the sputum; in the advanced form we should never make a diagnosis of tuberculosis unless tubercle bacilli are found. A word may be said as to verifying a positive result. Of the two cases in which we erred, one error was due to some mistake in the examination of the sputum. A positive result was reported but not confirmed. Either the specimens of sputum or the slides were mixed and a wrong report sent for that patient. There is a rule in the clinic that a positive diagnosis is not to be made on the result of one sputum examination. It must be confirmed. In this case it was not, and that led to error. Had we watched this point more carefully, we should not have had this error in the necropsy series. A positive sputum report is naturally regarded as giving a definite diagnosis; but it is easy to be careless and, in many laboratories, the slides are handled in such a way that a mistake is easily made. This is a point to which more attention should be given. Its possibility is suggested when patients come with a history of one positive and many negative examinations.

It will be noted that there is no mention of the mycotic infections. We have had these in mind as a possible source of error, but no instance occurred in the series so far as we could discover. In this connection, one comment may be made on the possibility of finding streptothrix which has come from the mouth. If a careful effort is made to clean the mouth thoroughly, the frequency of the occurrence of streptothrix in the sputum will be found to undergo a striking reduction.

The clinical errors in this series could have been prevented by more care and thoroughness. Some of the mistakes, as, for example, those in regard to the recognition of syphilis of the lung, or aneurysm, are readily made and, perhaps, not easy to avoid in the case of a man in general practice; but this cannot be said of the majority of mistakes. There is little excuse for the error made in diagnosing chronic car-

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diac disease or bronchitis as advanced pulmonary tuberculosis. One of our own errors was made through lack of attention in making sure that our rules as to sputum examinations were followed.

There is a lesson for sanatorium physicians. A number of these patients had been in tuberculosis sanatoriums and always regarded as tuberculous. This was the case with those patients, particularly, who had bronchiectasis. Some of them stated that a sputum examination had not been made, which is difficult to believe, but is supported by the fact that an incorrect diagnosis was made. In any institution dealing with patients with chronic disease, it requires constant effort not to rest content with a diagnosis once made. We are too likely to accept a diagnosis of advanced pulmonary tuberculosis as one not requiring special care to make, and, when once made, not needing revision. How many patients there are today wrongly regarded as having advanced tuberculosis, no one can say; but they are not few.

## CONCLUSIONS

1. In a series of 1,200 consecutive cases admitted as advanced pulmonary tuberculosis, seventy-two (6 per cent.) were found to be incorrectly diagnosed.

2. Among 134 cases coming to necropsy, in seven (5.2 per cent.) the diagnosis with which the patients came to the hospital was incorrect, and in two of them the diagnosis was not corrected.

3. The conditions most frequently diagnosed incorrectly were: cardiac and cardiorenal disease (nineteen cases), chronic inflammatory conditions (nine cases), bronchiectasis (eight cases), pulmonary abscess (eight cases), and chronic bronchitis and emphysema (six cases).

4. The diagnosis of advanced pulmonary tuberculosis should not be made unless tubercle bacilli are found in the sputum on at least two examinations.

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5. Physicians in tuberculosis sanatoriums and hospitals should take particular care to verify the diagnosis in all patients sent with a diagnosis of advanced pulmonary tuberculosis.

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## ABSTRACT OF DISCUSSION

DR. LEO KESSEL, New York City: It is difficult for most of us to understand that mistakes in diagnoses in patients supposed to be suffering from advanced pulmonary tuberculosis are made quite frequently. So much emphasis has been laid on the diagnosis of early tuberculosis that we have lost sight of the fact that it is almost as easy to err in the advanced cases. Unless you have seen a large series of heart and lung cases you will be unable to realize the frequency with which incorrect diagnoses are made. One of the important features on which stress should be laid is in regard to those nontuberculous respiratory infections. Patients suffering from such an infection usually apply for admission to hospitals in January, February and March. Ordinarily, they feel quite well during the summer. Their symptoms are of such a character that they are readily classified as tubercular patients, and we have seen quite a few who have spent many months at sanatoriums for tuberculosis. They usually complain of cough, profuse expectoration, dyspnea, sticking pains in the chetst, occasional hemoptysis, and a variable amount of weakness. Their physical signs are almost always located at the base, more frequently the left base, and they manifest themselves in dulness, crepitation, and diminished breathing. The sputum frequently contains pneumococci and streptococci, and, less commonly, the other organisms which we are accustomed to find in pulmonary conditions, but never tubercle bacilli. Dr. Funk called attention to the importance of recognizing syphilis of the lung as one of the possible factors in these cases with extensive physical signs in the chest and without tubercle bacilli in the sputum. I agree with him that it has become a fad to diagnose pulmonary cases as syphilis of the lung. I doubt whether we see more than two annually at Bellevue Hospital. We have had a large series of cardiorenal cases in which the mistaken diagnosis of pulmonary tuberculosis had been made. Quite a number of these patients have aneurysms. These aneurysms cause pressure on a bronchus, with cough, bloody expectoration, and fever, so that unless care is exercised a mistake in diagnosis is easily made. There are two other errors in diagnosis in these patients which arise not uncommonly. One is mistaking primary and secondary neoplasms of the lung for advanced pulmonary tuberculosis; the other is the overlooking of the complications of bronchopneumonia and

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lobar pneumonia which occur particularly in the pleura, and labeling these cases pulmonary tuberculosis. This mistake has been made during the past winter quite a number of times in cases of interlobar effusions. The point which I wish to emphasize is the importance of greater care in diagnosis with regard to those patients who present extensive physical signs in the chests and the sputum free from tubercle bacilli.

Dr. John A. Lichty, Pittsburgh: It is difficult to differentiate between chronic pulmonary tuberculosis and bronchiectasis, and mistakes in diagnosis are frequently made. The after effect of influenza on the lung is such as to simulate pulmonary tuberculosis. This must be borne in mind. Since this epidemic we have all had experience with cases which we would ordinarily diagnose as tuberculosis but which we now diagnose as influenzal lungs. Dr. Roland G. Curtis gave a series of lectures at Philadelphia in 1894 and 1895 in which he spoke of nonbacillary phthisis. Then we were not in the psychologic mood to accept that diagnosis because it was just after we had learned to stain the tubercle bacillus. Today we have exactly the same class of cases following this epidemic as followed the epidemic of 1889.

Dr. F. M. Pottenger, Monrovia, Calif.: We are missing tuberculosis ten times more often than we are diagnosing other diseases as tuberculosis. It is difficult to determine by clinical examination whether a given pathology is the result of an influenzal pneumonia or whether it is a tuberculous process. Latent tuberculous foci are found in all parts of the lungs and may be lighted into activity through influenza. Most patients who are suffering from postinfluenzal, nontuberculous disease have the lesions at the base. But the fact that the lesion is at the base does not indicate that it is of this character because latent tuberculous foci at the base may have been relighted into activity as a result of influenzal infection. The apex is the classical location for tuberculosis, but influenzal pneumonia also, at times, affects the apex and leaves a pathology which can not be readily differentiated from a tuberculous process. I see many cases of bronchiectasis and lung abscess which have been diagnosed as tuberculosis. The fact that bacilli are not found does not mean that the process is not of a tuberculous nature. Chronic tuberculous cavities may loose their bacilli for months at a time, but still many of them will throw off bacilli intermittently. This is particularly likely to occur when patients suffer from colds or bronchitis. A point in differentiating tuberculosis with cavity from abscess and bronchiectasis is furnished by the character of the lesion. A tuberculous cavity is situated in an area surrounded by infiltration and numberous foci of necrosis. These conditions favor percussion dulness and the presence of râles.

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Pulmonary abscess, on the other hand, is, as a rule, a single focus in tissue which is not surrounded by widespread infiltration and necrotic foci. Consequently, the condition which favors dulness and the presence of râles is not present. Bronchiectasis, being a disease of the bronchus with thickened walls, likewise does not favor dulness or the presence of large numbers of râles. Therefore, the presence of bronchiectasis and pulmonary abscess may be suspected on a clinical history of cough and free expectoration with a paucity of signs on physical examination. However, the treatment of all of these cases, whether tuberculous or not, is, in general, the same.

DR. A. G. SHORTLE, Albuquerque, N. M.: I want to call attention to the importance of a Wassermann test in the diagnosis of chest cases. In my own work I found about 10 per cent positives. I then compiled results of quite a number of other workers, and they averaged about 7.5 per cent. During the war I had to neglect this routine Wassermann, making Wassermanns only when the history or some uncertainty made it desirable, and I got only 3.5 per cent. positives. Four out of 1,300 cases is a very small percentage. If Dr. Funk had made routine Wassermanns he would have found that 7 or 8 per cent. had syphilis of the lung or syphilis as a complication.

DR. ELMER F. FUNK, Philadelphia: In regard to those cases which give a history that following influenza there have persisted for a varying time cough and expectoration with lowered weight and strength, we are going to be somewhat at a loss to separate the group which is simply postinfluenzal from that which is the result of a reactivation of an old tuberculous lesion in the lung. The roentgen ray does not help us much in the differentiation. We have frequently received from the roentgenologist some such report as this: "There is a definite shadow which I would be inclined to think was tuberculous. If, however, the man has recently had influenza it may simply be due to that cause." Under appropriate treatment, and by this I mean the usual rest, fresh air, good food and judicious application of drugs as prescribed in tuberculosis, the postinfluenzal respiratory lesions usually clear up promptly. In a certain number, however, and I refer in particular to the postinfluenzal bronchiectases, the cough and expectoration may persist with varying intensity for years. We would make fewer mistakes in the diagnosis of advanced tuberculosis if we kept in mind three simple rules: (1) Purulent sputum which does not contain tubercle bacilli is not due to tuberculosis; (2) marked symptoms suggesting tuberculosis and definite signs of an advanced pulmonary lesion without tubercle bacilli in the sputum is probably due to some other cause than tuberculosis, and (3) when a patient shows abnormal physical signs

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at the base with the apices clear, the possibility of tuberculosis is extremely unlikely. In 134 consecutive necropsies on patients with tuberculosis, we have never yet found a primary basal case, and in every one showing evidence of tuberculosis of the lower lobes one could trace out the old lesion to the apex. To repeat, when you get a basal lesion with the apex clear it is a pretty good evidence that it is not tuberculosis. On the other hand, apical lesions are not invariably tuberculous. Postinfluenzal catarrhs are frequently apical. Dr. McCrae and I reported before this section several years ago the case of a patient with nontuberculous apical bronchiectasis who was in several sanatoriums with the diagnosis of advanced tuberculosis. Every now and then malignant disease of the lung, either primary or secondary, will involve only the top of the lung. In these patients the symptoms and signs may suggest tuberculosis, but a careful consideration of the history, the unilateral character of perhaps an extensive lesion, and the persistent absence of tubercle bacilli in the sputum will exclude tuberculosis. One cannot emphasize too strongly the value of the roentgen ray in excluding those cases which ape tuberculosis clinically, but in which the symptoms and signs are due to various other causes such as foreign body, aortic aneurysm, malignant growth and deep seated loculated empyemas.

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# THE CORPS AND THE ARMY

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History repeats itself in generality rather than in detail. War recurs, but methods of warfare change, and so the detail of medical problems in modern war vary with the change of weapons and methods. By meeting these detailed changes successfully we achieve the unchanging basic object of the medical department—that of relieving suffering and maintaining the numerical strength and fitness of the forces.

What new details of medical nature did this war present, and how were they met by the medical organization in the Army and the corps?

The problem had three main branches: (1) transportation; (2) hospitalization, and (3) professional service—or the care and treatment of wounded. I shall consider only the last of the three.

The problem of sorting patients who arrived at the receiving tent of the divisional field hospital, you all know well. Probably in no busy city hospital of a great city was there ever found such a variety of disorders in such constant and overwhelming inflow; for in addition to the wounds there appeared, in great variation of expression, the interesting phenomenon of surgical shock. This profound physiologic disturbance, the cause of death in many cases in which the traumatism itself did not possess a fatal potentiality, was a medical problem of great importance. Besides these, the gassed, the exhausted and the sick offered unusual opportunities for differential diagnosis. War neurosis, effort syndrome and malingering completed an already confused mass of symptomatology.

Obviously, the kind of medical officer primarily required in such a field triage is an experienced clini-

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cian. And there should be available, likewise, the advice and assistance of experienced men in each of the domains invaded by modern warfare, gas, neurology and surgery.

About five or six years ago it was found necessary, because of many costly errors in diagnosis, to establish a special corps of experienced doctors to serve in the receiving ward, or triage, of Bellevue Hospital. The grave responsibility of forming correct clinical judgments with the necessary speed was found to be too heavy for the young ambulance surgeon. The qualification needed in the difficult receiving-ward position, more than any other, was experience. But we were a green army and did not know about the difficulties of triaging.

It was, as every one knows, the more usual thing in our triages to find any type of medical officer in charge, often the youngest lieutenant. In several instances the triaging officer was either a nose and throat specialist or an oculist. One cannot speak too highly of the way in which these men worked; but, obviously, the responsibility thrown on them was unjust, and their special services were also withheld from those who needed them most.

The next place in which experienced counsel was needed to assist the frequently undermanned staff was in the advanced corps field hospital—that new departure in field hospitalization. These hospitals were borrowed from replacement divisions and established near corps headquarters to care for the sick who trickled back with the wounded, shocked and gassed. They served the double purpose of preventing, in the first place, many secondary respiratory infections due to the crowding in evacuation hospitals and trains; and, in the second place, of returning men more promptly to the fighting front than had previously been possible.

Finally, in the evacuation hospital, which actually was a large secondary triage, there developed a most active and varied sort of medical service, which

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demanded the guidance of a well trained and experienced internist, in addition to the other special branches.

Some of the evacuation hospitals possessed such a man, but not all. Furthermore, not infrequently, problems developed in the various special hospitals serving the Army and the Corps, which demanded the attention of one or other of the consultant group representing some other branch than that of the given hospital.

To meet this urgent demand for the advice and assistance in purely professional matters, both in the combat area (where it was soon realized that what may be termed medical problems were as numerous and perplexing as the surgical), and also throughout the service of supply, a special department was arranged, known as the professional services. It was an attempt to solve quickly a situation which, for some cause, had not been fully anticipated. For one reason or another, which it is not necessary to discuss now, this professional services group found the road to useful service beset with many a thorn. For brevity's sake, it may be said that coordination between this new branch and the existing organization, as called for in the Manual of the Medical Department, was incomplete.

The new group was unfortunately given the name of "consultant," and the "consultants" were sent out to the Army corps to act in an advisory capacity, but utterly without authority. Their reception by corps and division surgeons varied all the way from cordial cooperation to definite antagonism. Obviously, something was wrong with a system of military affairs the success of which depended on personal disposition and relationship.

The consultants in medicine attached to the corps were expected, during combat, to oversee the work of surgical shock carried on in the field hospitals of divisions operating in the corps, and to act as epidemiologists in the sector. At the outset there was no well-defined policy as to the treatment of surgical shock and

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emergency surgery, nor any means of carrying out epidemiologic activities. But each corps consultant worked out his own ideas, to the best of his ability, on the soundest principles he knew. This naturally resulted in great confusion and annoyance to the division surgeons when, as often happened, their divisions were moved rapidly from one corps to another, and the methods of the corps varied. Nevertheless, it became apparent that, with a little tact, the corps consultant could be of great usefulness. For, besides assisting directly at the field hospitals in the treatment of surgical shock and gassed patients, he could direct the work of the corps field hospitals for the acutely sick.

### CONCLUSIONS

There is perfectly clearly a need for experienced men in the triage, advanced corps field hospitals and evacuation hospitals. Their work should be properly coordinated with the other parts of the medical organization; but they must be essential elements of the medical personnel, clothed with authority commensurate with the responsibilities of their work. It is believed that there should be formed two distinct but well balanced and interlocking parts to the military medical service of the Army and the corps—one administrative, the other charged with the purely professional service of caring for the sick and wounded.

## ABSTRACT OF DISCUSSION

Dr. Homer F. Swift, New York: For the most effectual utilization of professional services there must be close cooperation between the chief surgeon, no matter at what point he may be, and the consultant. The consultant, whether in the division, the corps, or the army, who is not the assistant in fact as well as in name, will lose most of his effectiveness. This depends on the chief surgeon's ability to utilize him, as well as on his willingness to cooperate. Obviously, in any organization there must be some one person who is responsible for the entire work. In the medical corps it is the chief surgeon of any particular unit. If he delegates the supervision of their particular line of work to the various consultants, they are as much a part of his

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organization as the medical supply officer. In the different grades the functions of the consultants are somewhat different. As Dr. Draper has noted, it is essential that there be a definite, uniform policy in the professional care of sick and wounded throughout the entire army, because divisions are moved quickly from one corps to another, as well as from one army to another. The army consultant should, from conference with the chief consultant of the expeditionary forces, be able to suggest the policies that are to be carried out in the divisions and in the evacuation hospitals, and to supervise the work in the evacuation hospitals. The corps consultant should supervise the work in the division, acting as an instructor and as an inspector. He should establish the liaison between the division field hospitals, the chief surgeon of the corps and the army consultant. Where there are corps field hospitals he should assume direct supervision of the professional work in them. If there are divisional consultants they should assume the direct care of the patients as long as they are in the division field hospitals, and supervise the work in the forward areas. With such a distribution and delegation of responsibility, and with frequent conferences between the various consultants and chief surgeons, there will be a uniformity in the care of sick and wounded that cannot be obtained in any other way.

Dr. William S. Thayer, Baltimore: We must remember that the whole development of this system of professional services was in a fluid state. It was begun in November or December, 1917, to meet existing conditions. It was changing and developing constantly. The main difficulties lay in the fact that it was a superstructure that had not yet become fixed or become settled or developed in the army. One point has been brought out that appeals to me very strongly, and that is the necessity in the future of some proper separation of or differentiation of administrative from professional duties, and the recognition of the circumstance that from the front to the rear it is necessary to have some form of recognized professional supervision, there should be someone who is the recognized professional director, advisor or consultant, call him what you will. That, I think, the experiences of the war, show very definitely. It has been mentioned by some that one of the difficulties was that the responsibility was not easily fixed; it was not easily clear from where authority was derived. All that was in large part dependent on or due to the circumstances that the whole scheme was in the process of development, and while those of us nearer headquarters had very definite ideas as to what it meant, we did not always realize or remember that nearer the front some of the officers scarcely knew of the existence of the superstructure. Some definite plan should be devised to meet such problems, if they arise in the future, as some day they will arise.

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# THE WORK OF THE GROUP CONSULTANT

JOSEPH SAILER, M.D.
PHILADELPHIA

The center consultants in the American Expeditionary Forces commenced their work late in the period of the war. The largest group did not reach France until September 3, and there was a delay of more than a month before all of them were distributed to the centers in which they were to work. Their duties, unfortunately, were ill defined, and their actual authority was very slight. In some regions there was considerable jealousy of their activity on the part of the commanding officers of the centers and of the different hospitals; but on the whole they experienced very little difficulty. This, I believe, was largely a matter of personality; for without definite powers, their functions were chiefly suggestive.

In different centers the work necessarily varied considerably, and the problems presented were often very different. There were two rather distinct types of hospital: those constructed for temporary use practically on the plan of the base hospitals at the various camps in the United States, and those occupying hotels in some resort district. It was to a center of the latter type that I was attached. The nature of my work was largely governed by the difficulties experienced in turning hotels into satisfactory hospitals. The first problem to be met was devising some method for the more efficient care of the patients. Primarily, segregation of the different groups of cases seemed the most important initial procedure. In spite of the fact that the French had very generously turned over some eighty hotels to the American government at Vichy, the num-

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ber of patients constantly exceeded the normal capacity until after the armistice had been signed. This led to overcrowding to a very serious degree and all the difficulties that go with such a condition.

When a troop train arrived, patients were assigned to the vacant beds without any particular reference to diagnosis. As a result, it often happened that patients suffering with pneumonia were placed in the same wards with patients suffering from gas poisoning or influenza and also with surgical cases. Cubicles were practically unknown, and no attempt whatever had been made to prevent cross infection.

### THE ESTABLISHMENT OF A PNEUMONIA HOSPITAL

The commanding officers at the different base hospitals were at first distinctly opposed to have either pneumonia wards, or what seemed more desirable, a hotel devoted solely to pneumonia cases. To the former they made the objection that the ward might not always be full or that there might be an overflow. In the former case it would crowd the other parts of the hospital, and in the latter case no advantage would be gained, as the surplus cases would of necessity be placed in other wards. To the plan of a pneumonia hospital they objected that their ward surgeons should have the opportunity to see and treat cases of pneumonia. Through the aid of Colonel Webb, the commanding officer of the center, the latter plan was adopted, and no difficulty whatever ensued. The ward surgeons were, on the whole, rather glad to be relieved of any added work, and all recognized the advantages to the patients.

One hotel was reserved for this purpose. The active cases were placed on the first floor, the second floor was reserved for the nurses and officers, and the third, fourth, fifth and sixth floors for the convalescents. This enabled us to hold the patients until there was

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little danger of sending carriers out among the other men.

THE MANAGEMENT OF OTHER DISEASES

The management of influenza was less successful. It was impossible to secure a hotel for these cases; but it was possible to set aside certain wards, and the men were kept fairly well isolated. The subsidence of the epidemic relieved us of all anxiety.

Of typhoid fever, we had only a small number of cases. These patients were isolated in a ward set apart, and another ward was provided for typhoid suspects. The same precautions were used, and no epidemic occurred. Two cases that proved to be typhoid came to necropsy with other diagnoses. In one, the clinicians had regarded the case as typhoid from the beginning; but the laboratory failed to confirm this diagnosis in numerous blood and stool cultures. The second patient was admitted to the hospital moribund and died a few hours later. No attempt at diagnosis was made.

One hotel had been set aside for contagious diseases, and a second hotel next door was subsequently acquired. Separate floors were set aside for the diseases we encountered, and no difficulty was experienced in handling the considerable number that were found. Diphtheria caused us a good deal of anxiety. From one hotel we had from four to eight diphtheria patients sent over to the contagious ward every day. The commanding officer of the base hospital in charge of this hotel insisted on making light of the condition, and was most unwilling to take any precautions. Fortunately Colonel Webb became aroused to the condition after a commission had been appointed to study the situation and had made its report to him, and we were able to carry out measures designed to check the epidemic. Every person in this hotel was masked. Outsiders were not permitted to enter, and cultures were taken from every one at intervals of twelve hours until three cul-

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tures had been taken in order to detect any possible carriers. All together, twenty-eight carriers were found and removed to the rooms reserved for diphtheria carriers in the auxiliary contagious ward. This stopped the epidemic. In the ensuing three weeks only two cases were reported from this hotel. It was very easily accomplished and did not disturb either the work of the center or the work of that particular base hospital appreciably.

Some interest was aroused by the discovery, in the diphtheria ward, of two wounds infected by the diphtheria bacillus. Both healed very rapidly after the administration of diphtheria antitoxin. As a result, when cultures were taken in the infected hotel, all the open wounds were also cultivated; but no diphtheria infections were found, and we concluded that this is a comparatively rare complication even when diphtheria bacilli are prevalent.

Mumps and measles were comparatively rare. One typical case of cerebral mumps corresponding closely to Trousseau's description excited much interest. The patient made a satisfactory recovery.

Another interesting feature was the management of diarrhea. As nearly as we could determine, this was due to a chronic irritation of the colon; but the cause could not be found. At least there were no amebas, no reaction to dysentery bacilli, and no evidence of chronic pancreatitis or achylia or intestinal parasites. It seemed to be largely dietetic. Ultimately we decided to keep these patients in one hotel. They were all placed on a careful diet increasing in quantity as improvement occurred, and they rapidly recovered. It was almost impossible to keep them on a diet in the wards with other patients, owing to the generosity of their comrades.

### PROBLEMS OF OUTLYING HOSPITALS

The problems presented by the outlying hospitals of my district were not very different from those in

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Vichy. In one district the commingling of different types of disease and surgical cases was very great. The commanding officer again felt that any attempt at classification would be futile on account of their overcrowded condition; but with the enthusiastic support of the chief of the medical service we soon had separate wards for pneumonia, influenza and typhoid fever, besides a well managed ward for the severe forms of contagious diseases. Instead of disturbing, this greatly facilitated the work of the hospital.

METHOD OF MAKING USE OF CLINICAL OBSERVATIONS

The peculiar opportunity of the group consultant consisted in the vast amount of clinical material under observation. In order to utilize this it was necessary to arouse the interest of the chiefs of the service and of the ward surgeons. For this purpose various methods were tried, but only two proved at all successful. A medical society was organized that met once a week before which cases and specimens were presented and reports read. A satisfactory voluntary attendance was finally secured, and the work was excellent. The discussions particularly were sometimes very vigorous. Great assistance was rendered in this work by Captain Martland, who was in charge of the central laboratory. The ward surgeons were encouraged to tabulate the records of the various groups of cases. The study of cases of thoracic wounds by Lieutenant McGuire[1] of Base 1 was notable.

The numerous and varied amusements necessary in such a large center were unfortunately somewhat distracting. Among the interesting clinical observations I shall mention only two. Of sixty-six cases of pneumonia, all of which had complete necropsies, five showed unsuspected hemorrhages on the base of the pons. All of the sixty-six patients showed purulent

1. McGuire, W. A.: Hemothorax Following Gunshot Injuries of the Chest, J. A. M. A. 72: 1269 (May 3) 1919.

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infection of practically all of the accessory sinuses of the nose. Such complete necropsies of the head in pneumonia have not, I believe, hitherto been performed in such a large number of cases. One patient in Base 19, with massive edema of the head, arms and upper thorax, with no shadow of aneurysm but a positive Wassermann reaction, recovered completely with antisyphilitic treatment, showing that this syndrome is not due solely to thoracic aneurysm perforating the descending cava.

### DEFECTS IN THE ARMY MEDICAL CORPS

While the base hospitals in clinical personnel and equipment compared not unfavorably with the better civil hospitals in America, the care of the sick fell short of what we desired to achieve. For this there are many, and I believe, adequate explanations; but I am less interested in stating excuses than in attempting to suggest remedies in order that we may be better prepared should a similar occasion arise in the future.

To my mind, the fundamental defect in the Army Medical Corps, as at present organized, is the exaltation of the administrative over the clinical work. This results unfortunately in several ways. Some good clinicians were persuaded to undertake the administrative work because it offered more rapid promotion and the satisfaction of authority, and were thus unable to do the more useful work. Some officers who did not understand clinical medicine were given autocratic authority and hampered the work of the clinicians subject to their orders. I have come to believe that in the army a skilful surgeon, internist or specialist is more valuable to the wounded or sick soldier in his particular line than he is if he devotes himself to the work done satisfactorily in civil life by the hospital superintendent, and that adequate encouragement to clinical work in the Army Medical Corps would add considerably to its efficiency.

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# THE WORK OF THE GROUP CONSULTANT IN FRANCE

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The position of medical consultant was not created until late in the summer of 1918. It had long been recognized, however, that there was need of a closer relationship between professional services of the different cantonment hospitals, and for this purpose, an arrangement was made for the exchange of visits between chiefs of medical services. These visits proved to be of such value that the position of consultant was created to establish a similar liaison between the hospital services overseas.

Thus the appointment of consultants in the American Expeditionary Forces with headquarters at Neufchateau was an innovation and an experiment. The medical consultants were assigned to duty by Brig.-Gen. W. S. Thayer with the injunction to make good and thereby justify their existence. All these officers had already served as medical chiefs in hospitals at home and were therefore familiar with military duties.

The assignments embraced both base and camp hospitals, so that the consultants had a variety of experiences, but in the main the work developed along the following lines.

1. Consultation over individual cases with the medical chief and his staff. This made up a part of every day's work. Frequent visits of the consultant to many different hospitals enabled him to keep in touch with new or unusual conditions and to pass along helpful information concerning diagnosis and treatment.

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2. Standardization and improvement of quarantine. This was an important feature of the service. The consultant, in conference with the medical and laboratory chiefs, drew up regulations for the separation of gas, influenza and pneumonia cases; for the prevention of cross infections in the wards; for determining the period of quarantine, etc. Much of the success of the consultant depended on his ability to cooperate with the commanding officer. As a rule this officer welcomed these recommendations and was disposed to enforce them.

3. Unification of the medical services and promotion of an esprit de corps in the medical personnel. In the larger centers there were many hospital units working side by side on similar problems and yet with an amazing ignorance of each other's activities. The men were not acquainted with their neighbors on account of the exacting demands of the service; but it was obvious that an interchange of experiences and ideas would be of mutual benefit. Medical societies were organized with a weekly meeting devoted to clinical medicine and another to pathologic demonstrations. As a result the general medical services were brought together and a scientific interest stimulated that was immediately reflected in the quality of all the medical work.

4. Monthly reports to the chief consultant on the incidence of disease and the personnel. Special reports and recommendations were also made at irregular intervals on epidemics or conditions of unusual interest.

### MEDICAL PROBLEMS OF SPECIAL INTEREST

1. The gas patients received their first treatment in the acute stages in the triage, evacuation and special gas hospitals. The phosgen and chlorin victims had usually passed the dangerous period when they arrived at the base hospitals. The mustard gas patients, however, developed serious complications in the base areas,

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chiefly from purulent bronchitis and bronchopneumonias. The great problem with these patients was to prevent secondary infections.

2. Gas edema or subcutaneous emphysema occuring in unwounded cases of pneumonia presented a unique clinical picture. Eight cases occurred at La Valdahon and three at Allerey. In most of the patients the emphysema started in the neck and spread to the face and arms, chest and abdomen and often to the scrotum. About one half of them died. In the sputum, blood and tissues in one case, a gas-producing bacillus was obtained. It is hoped that this unusual complication will be fully reported by the physicians in charge.

3. Influenza was not a clean-cut disease, consequently the statistics on influenza should be interpreted very liberally. The simple cases ended in recovery, while those contracting secondary infections frequently developed bronchopneumonia.

4. Gross infections and multiple infections of the respiratory tract were very prevalent in gas, pneumonia and "flu" patients. It was generally believed that the prevention of such multiple infections would greatly reduce the frequency of pneumonia and cut down the mortality percentage. Great efforts were made in most of the hospitals to accomplish not only ward quarantine but also individual isolation by means of sheet cubicles and by the use of gauze face masks on the part of ward surgeons, nurses, attendants and patients. The difficulties of enforcing these measures were considerable, but every week showed a marked improvement in the technic.

### RESPIRATORY INFECTIONS AND TRANSPORTATION

An unlimited and comparatively undeveloped field of usefulness is open to the consultant, in the control of respiratory infections among the sick and wounded

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during the period of transportation from the front to the base hospital. On the hospital trains, wounded men and susceptible gas patients were often huddled together in the same compartments with influenza patients. Many soldiers acquired respiratory infections on the trains. One of the great opportunities for preventive medicine is in careful segregation and isolation. In some centers, all ambulance cases were marked and plans had been formulated to carry out similar protection with masks and curtains on hospital trains when the armistice was declared.

It is only fair to say, in conclusion, that considering the shortage of physicians and the enormous number of patients, the character of the medical work in the American Expeditionary Forces was altogether a credit to the profession.

## ABSTRACT OF DISCUSSION

### ON PAPERS OF DRS. CAPPS AND SAILER

DR. R. T. WOODYATT, Chicago: Dr. Pincoffs brought out and Dr. Thayer emphasized a point of great importance in connection with the regiment and division in their relationship to the medical department, namely, the performance of a large amount of administrative work by men who were trained essentially as physicians. Of course the training of a physician should not stop his training as an administrative man, but the point made was that an accomplished special training was wasted for the development of an administrative training at a late hour. I am not able to speak about the problems at the base hospitals in France with the authority of men who were there, but I think the same principles prevail all the way back to the base hospitals in this country, and I should like to point out the fact that a great deal of time and energy is wasted by medical officers who must perform administrative duties without the means at their disposal to organize their work so that nonmedical duties may be performed by nonmedical men. That is to say, it is a purely medical personnel which is at the disposal of a chief of service in a base hospital. There is a great deal of administrative work to be done. The medical officer should do it or see that it is done, and if he does not, he should be replaced. Nevertheless the service chief in charge of many administrative details should have at his disposal a certain number of sanitary corps men to perform those purely administrative functions which do not

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require the services of a doctor. Otherwise, he will waste professional training and get poor administration.

Dr. Robert H. Halsey, New York: Let me call attention to one phase which is common to all consultants. Whether the consultant be attached to the division, corps or army, he depends on the authority of the commanding general, through the chief surgeon, for the execution of his recommendations, and in base hospitals or hospital centers the consultant must obtain the authority and cooperation of the commanding officer of the unit or group. The efficiency of the consultant depends, therefore, on his ability to obtain the authority of the commanding general through the chief surgeon with whom he is associated. In some instances the chief surgeon of the division or corps did not have the cooperation of the commanding general, and he would have to plead for the authority or order the need of which was perfectly evident to the chief surgeon and the consultant. In other cases the consultant would have to argue with the chief surgeon in order to persuade him of the necessity of the solution of the problem presented. The same state of affairs existed in base hospitals and hospital centers. It is essential that the consultant be a patient, tactful teacher of his chief surgeon and, most important, that he be an enthusiastic student of the problems which his chief surgeon has to solve. It must not be forgotten that the military machine is for fighting and the function of the medical officer is to care for the property, or men, of which the machine is made, but the director, or commanding general, of the machine must at all times be the sole authority as to how the property shall be cared for. The efficiency of the consultant will depend, in the last analysis, on the manner in which he gains and keeps the authority and cooperation of his chief. No system can be devised from which can be eliminated this element of tactful interchange of knowledge. The moment a consultant attempts to put into execution orders depending on his own authority, at that moment he weakens or loses his influence. The consultant must not be ignorant of nor disregard the administrative portion of his army work; he must at all times keep his chief accurately posted as to conditions and his recommendations for the solution of the problems, and he must keep himself thoroughly informed as to the difficulties of the administrative work of his chief. The cordial interchange of knowledge with mutual, sympathetic cooperation is the real problem of the consultant to whatever unit he may be assigned.

Dr. William Darrach, New York: The problem of the consultant is a real one, if the position is to be made use of in the future. The difficulties met with in the A. E. F. were many, but a working plan was being evolved gradually and an excellent example of this was shown in the third army.

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The chief surgeon, Colonel Grissinger, recognized the possibilities of the consulting staff and made use of them both in the first corps and later in the army of occupation. The organization of his office was an excellent example of coordination between the administrative and the professional branches. In the former department men were in charge, respectively, of (a) hospitalization, (b) supplies, (c) personnel, (d) transportation, and (e) executive. On the professional side there were consultants in (a) surgery, (b) medicine, (c) skin and venereal diseases, (d) laboratories, (e) ear, nose and throat, (f) eye, (g) orthopedics, (h) psychiatry and neurology, (i) sanitation and (j) epidemiology. Of this latter group almost every man had had previous experience in both base and divisional work and many of them had served as battalion medical officers. That is, they had learned the different aspects of the army medical work. The chief criticism which can be made against us as consultants is, I think, that many of us did not take the pains to learn practically the administrative side of the problems presented. This knowledge was necessary for proper cooperation and correlation.

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# SURVEY OF THE EPIDEMIC OF INFLUENZA IN THE AMERICAN EXPEDITIONARY FORCES

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The origin of the epidemic of influenza and its invasion of Europe is still obscure; but it is certain that influenza existed in epidemic form in Spain, France and possibly England, early in 1918, and it may even have been present in endemic form during the previous year. By May, 1918, there was in Spain a widespread epidemic which received much publicity. In April or May there were local outbreaks of the disease, according to the report of the British commission, in the first and second British armies, and by the late spring influenza was present in epidemic form, not only in Spain and in France, affecting both the British and the American armies, but also in Italy, Belgium, Austria and Germany.

Until the complete official reports of the incidence of influenza in the American Expeditionary Forces are made available and the statistics of the epidemic published, it is impossible to give figures or similar accurate data which bear on the epidemiology of this disease among our own troops in France. A paper by Dr. Ward J. MacNeal,[1] assistant to the director of laboratories and infectious disease, American Expeditionary Forces, which recently appeared, is an official report on the epidemic, and contains considerable data of interest. Dr. MacNeal kindly allowed me to see the manuscript and to quote from it. Much of what I have

1. MacNeal, W. J.: The Influenza Epidemic of 1918 in the American Expeditionary Forces in France and England, Arch. Int. Med. **23**: 657 (June) 1919.

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to say, nevertheless, must be based on personal observations that are of necessity fragmentary and limited.

As one reviews the history of the epidemic in France, it may be divided into three fairly definite outbreaks with an intermediate and interesting phase bridging the gap between the second and third outbreaks.

The first outbreak appeared in April and May, 1918. It was comparatively mild and spoken of as "three day fever."

The second outbreak came in September and October, 1918, and formed a part of the terrible pandemic.

The third definite outbreak, according to recent reports, was much less serious, and occurred in January and February, 1919.

The fourth or intermediate phase was represented by scattered cases of influenza and great numbers of respiratory infections, either unaccompanied by pneumonia or associated with a pneumonia of the true lobar type. This phase followed the second great outbreak, and continued during the autumn and winter of 1918-1919.

### THE FIRST OUTBREAK

In the first outbreak the earliest recorded epidemic among the American Expeditionary Forces appeared, according to MacNeal, about April 1, 1918, at Rest Camp No. 4 in Base Section 2, near Bordeaux. It reached its height, April 22, and ceased, May 5. In May, 1918, a second outbreak of influenza was reported from the Quartier de Beaumont, Tours, which lasted from May 1 to May 24, and affected 117 persons. During this month another epidemic occurred at Chaumont, and from this time until July, more or less extensive epidemics appeared throughout the entire American Expeditionary Forces. The disease was likewise prevalent at this time among the French population and in parts of the British army. It is stated by the British commission[2] that in the first British

2. A Report on the Influenza Epidemic in the British Armies in France, 1918, by the Influenza Committee of the Advisory Board to the D. G. M. S., France, Brit. M. J. 2: 505 (Nov. 9) 1918.

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army, between May 18 and July 2, 36,473 cases of influenza were admitted to casualty clearing stations. It is obvious, therefore, that a widespread epidemic of influenza prevailed throughout France in the spring of 1918, reaching its height in May and June, and subsiding during July and August.

Influenza in this first outbreak was a comparatively mild disease. It was notable on account of the rapidity with which it spread through any organization, on account of the high morbidity rate, the short duration —from two to four days—and the rarity of complications.

The onset was sudden with fever, headache, prostration, pain in the eyeballs, pain in the back, soreness of the muscles, occasional abdominal pain, and rarely vomiting and diarrhea. Epistaxis was common. Occasionally a dryness of the throat with a harsh cough was described. In many cases there were conjunctivitis, slight suffusion of the face and some reddening of the fauces. Muscular tenderness and, more rarely, abdominal tenderness, were present. The fever lasted from two to four days, and convalescence was rapid and almost always uneventful. At this time serious involvement of the respiratory tract was so uncommon that this feature attracted little attention. Only the milder manifestations already mentioned were observed in most localities, though in a few places pulmonary involvement was referred to. In the first British army, it was estimated that in 1 per cent. of all cases coming to the casualty clearing stations, bronchopneumonia developed, from which 10 to 15 per cent. of the patients died. In one epidemic in the Eleventh Engineers, A. E. F., reported by Bradbury,[3] among 613 cases of influenza there were ten of severe laryngitis and four instances of bronchopneumonia, from which one of the patients died. It is clear, therefore, that while the first outbreak was generally of a mild character, the

3. Bradbury, S.: Am. J. M. Sc. **156**: 737 (Nov.) 1918.

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upper respiratory tract was not infrequently affected, and occasionally pneumonia recurred as a complication.

### THE SECOND OUTBREAK

The second and serious outbreak started during the latter part of August and early in September. In the early epidemics at this time the involvement of the respiratory tract first became alarming. Even by Aug. 18, 1918, in an epidemic at an artillery camp at Valdahom that has been carefully studied by Major Snow and Captain Alan M. Chesney, the disease in most of the 203 patients that were in the hospital presented symptoms of involvement of the upper respiratory tract. Many patients presented signs of the acute emphysema to which Torry and Grosh[4] have called particular attention in the cases in this country, while the development of pneumonia was not uncommon. Later with the assignment of fresh troops to this camp, the epidemics became much more serious in nature, owing to the frequency of pneumonia. A little later and during the early part of September, a similar but much larger epidemic took place in an artillery camp near Bordeaux, and though the cases of pneumonia were not very numerous, the mortality among these patients was high.

From the latter part of September until the last of October, the outbreak was at its height. This was approximately the same time that the influenza raged in America. The disease seems to have been the same in the two continents. If it were possible it would be of the greatest interest to compare the incidence of the disease and the morbidiy and mortality rate in the different units and organizations in the American Expeditionary Forces with the figures that have been collected from the camps in this country, for in America the troops were under fairly uniform conditions, whereas in France these varied tremendously. It is

4. Torrey, R. G., and Grosh, L. C.: Am. J. M. Sc. **157**: 170 (Feb.) 1919.

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fair to say, however, that the disease was present throughout France in epidemic form from the base ports to the fighting front. It prevailed particularly, however, first, among the troops at the base ports where during a part of the epidemic transports laden with infected troops were being landed; secondly, in those organizations that contained the largest numbers of replacement troops; and thirdly, in organizations that were being moved on troop trains, where the men were necessarily closely crowded.[5] The effect which exposure and cold had on the incidence of the disease and its share in determining the prevalence of pneumonia as a complication is not perfectly clear. Troops which were at the front were affected, but it seems highly probable that some organizations were much more severely affected than others which operated under very similar conditions. A study of carefully collected figures and an analysis of the exact conditions would be necessary before one could make any statement regarding this matter. It did seem clear, however, that crowding of influenza patients and their transportation on trains increased the incidence of pneumonia. It will undoubtedly be extremely difficult to determine the morbidity rate in the American Expeditionary Forces, but there is little reason to suppose that it will differ materially from the figures and variations that occurred in this country. An epidemic in one organization which was well separated from other American divisions affected about 20 per cent. of the men. It was further estimated that there were upwards of 1,000 cases of pneumonia, of which 30 per cent. were fatal. This is not very high as compared with the tables which Soper[6] gives for the camps in this country.

The symptomatology, the clinical course, the pathology and the bacteriology of the disease seem to have been in the main the same in France as they were in

5. Epidemic Influenza Among American Soldiers Abroad, Pub. Health Rep. **33**: 2035 (Nov. 22) 1918.

6. Soper, G. A.: The Pandemic in the Army Camps, J. A. M. A. **11**: 1899 (Dec. 7) 1918.

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this country, and it is, therefore, unnecessary to describe the clinical course which is familiar. At many hospitals that interesting complication, generalized subcutaneous emphysema, which has also been reported from camps in this country, was observed. The mortality in this group was high.

There is a second group of cases to which attention must be drawn. In these meningococci were obtained in culture from the sputum, the blood and in enormous quantities from the pneumonic lungs at necropsy; and it seems highly probable that the meningococcus played some part in the epidemic as a secondary invader and must be classed as such with the various types of pneumococcus, *Streptococcus hemolyticus* and *Staphylococcus aureus*. The etiology of the disease and, therefore, the position of the influenza bacillus in relation to it, are outside the present discussion. There is one other group of cases which was common in the American Expeditionary Forces, but to which reference has rarely been made. These are the cases of influenzal pneumonia running a prolonged or subacute course. In many of these patients, signs suggestive of lobular pneumonia with extensive bronchitis persisted for weeks and were frequently confused with tuberculosis. It is highly probable that some of these patients had developed bronchiectasis.

### OTHER OUTBREAKS

The second and terrible outbreak subsided during the early part of November. The third outbreak of influenza is reported as occurring in January and February of 1919; but since very few data are available concerning this epidemic, I shall not discuss it here.

Between the second and third true outbreaks there occurred what I have termed the fourth or intermediate phase of this epidemic; and though the infections during this period must be considered as a sequel to, rather than a part of, the influenza epidemic, they nevertheless have an important bearing.

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With the subsidence in November of the great outbreak, respiratory infections of all types became extremely common, and though it was difficult to differentiate many of these infections from influenza, the large majority did not conform to the clinical picture which had previously been familiar. Laryngitis, tracheitis and bronchitis were extremely common. As a rule, the infections ran a fairly prolonged course with slight initial fever of a day or two. True lobar pneumonia was not unusual. The lobular pneumonias were less common than before. Many of these patients had previously had an attack of typical influenza. There was a leukocytosis in some of the patients observed. Although it is difficult to come to any definite conclusion as to the position which this intermediate phase bears to the general epidemic, it seems highly probable that respiratory infections at this time were not all instances of influenza, but were usually due to the bacteria which had been secondary invaders in the original epidemic. As an explanation of this phase of the epidemic, therefore, one might suggest that the bacteria which as secondary invaders had been widely disseminated and had repeatedly passed through numerous susceptible hosts during the influenza epidemic, had acquired increased virulence and certainly wide distribution and were responsible in themselves for the great prevalence of more or less serious respiratory infections during the few months following the second serious outbreak of influenza.

## SUMMARY

It may be said that the influenza epidemic in the American Expeditionary Forces was characterized by three definite outbreaks or peaks: the first between April and July, 1918; the second in September and October, and the third in February and March, 1919. Between the second and third peaks there was an intermediate phase.

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The first outbreak was comparatively mild, and at this time involvement of the respiratory tract was unusual and attracted little attention. In the second outbreak, infection of the respiratory tract with the occurrence of lobular pneumonia became rapidly the predominant feature and was responsible for the high mortality.

These respiratory infections were often associated with the influenza bacillus, but were largely dependent on a secondary invasion of pneumococci, streptococci or, in some instances, even of meningococci.

Following the second peak, there came the intermediate phase during which time the variety of influenza observed in the early outbreaks was rarely seen but respiratory infections of all sorts were extremely prevalent, and one gained the impression that the organisms acting as secondary invaders during the epidemic proper were the primary causes of the bronchitis and pneumonia which was widespread at this time.

The character of the disease occurring in the second peak was the same as that which spread at the same time throughout the United States.

The influenza epidemic affected troops in all parts of France, but prevailed especially at the base ports, in those organizations containing the greatest number of fresh replacements, and in organizations in which the men were of necessity densely crowded together and subjected to excessive fatigue and exposure.

## ABSTRACT OF DISCUSSION

DR. ALBERT E. ROUSSEL, Philadelphia: I would like to ask Dr. Longcope what the mortality is in the uncomplicated influenza cases and by that I mean that bronchitis can be accepted in most of these cases as one of the necessary accompaniments of the respiratory tract, what the mortality in these uncomplicated cases is. In the experience of the two epidemics of influenza I think that the mortality of the influenza even of the more virulent type is as yet relatively small—not more than possibly 2, 3 or 4 per cent., that the

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greater mortality is due to complications often unrecognized so that I do not see why an attempt should not be made to differentiate from influenza proper and its complications. We do know that the pneumonic complications were of a particularly high rate of mortality. Another particular point of interest, I would like to know the proportion of bronchopneumonias to lobar pneumonias. Out of 186 cases of pneumonias in my service there were only seventeen cases of lobar pneumonia. And yet another question, I would like to know the number of cases of kidney involvement of various degrees. No less than 8 per cent. of these cases showed kidney involvement, probably 4 per cent. an acute nephritis.

Dr. Edwin R. Le Count, Chicago: It seems to me that some of the members might be interested to know that the first outbreak of influenza was in April, 1918. In Chicago the first postmortems were made by me in the first week of April, 1918. I think there may have been other cases in this country. We did not know at that time what we had. The lungs were full of hemorrhages. It was a reasonable thing to diagnose them as poliomyelitis and meningitis. I sent for my colleagues, Dr. Hektoen and Dr. Davis, to look over it as a new disease. Dr. Davis studied it bacteriologically. We sent everything worth while to a chemist and made histologic examinations, but it was not until the fall that we knew what we had, when we had cases duplicated.

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# NEPHRITIS IN THE SOLDIER*

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Two types of nephritis were encountered abroad in soldiers during the recent war. Chronic nephritis was a rare disease and can be dismissed without further discussion, as obviously it was to be excluded by a thorough routine physical examination of all recruits before enlistment. Acute nephritis was more common and a greater problem from the points of view of medical administration as a whole, and individual care of patients in particular.

Acute nephritis was not important in warfare until the present time with the exception of the Civil War, when there were approximately 14,000 cases. It was not prevalent in the Franco-Prussian War, the Chino-Japanese War, the Soudan War, the Spanish-American War, or in the South African War. After 1915, however, it appeared among British, French, Austrian and German troops. No figures have been published as to the frequency of the disease in the American Expeditionary Forces. Among the British in 1917 and 1918, as judged by the admissions in two typical general hospitals, it represented about 5 per cent. of all medical casualties sent from the line.

The type of acute nephritis was so well marked clinically that it came to be called "trench" or "war" nephritis to differentiate it from chronic nephritis or acute nephritis of other types. It had four outstanding clinical characteristics: acuteness of onset; the symptom of dyspnea apparently not of cardiac or mechan-

* From the Medical Division, United States Army Base Hospital 5.

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ical origin; the rapid development and disappearance of edema, and the suddenness and frequency of uremic manifestations. The disease affected all branches of the army and was prevalent among officers as well as men. The etiology was not discovered. Many theories as to the cause were put forward, such as exposure, unwholesome drinking water, lead, or the concentrated and high protein diet contained in rations. None were substantiated. No predisposing factors could be demonstrated.

Physical examination of patients with trench nephritis was essentially negative except for dyspnea, high blood pressure and general edema. The dyspnea was not associated with a marked hydrothorax or with cardiac dilatation. There was often a diffuse coexistent bronchitis without signs of consolidation. The blood pressure was sometimes normal or subnormal, but was usually 170 mm. or more. The edema was most marked in the extremities, over the sacrum and chest wall, and in the face. Edema of the genitals occurred with less regularity, and marked ascites was rare. Retinal hemorrhages were not often seen, and true albuminuric retinitis was unusual.

The course of the disease was that of a subacute nephritis tending to last for several months but not to develop into a chronic progressive nephritis. After a period of rest in bed the patient's fluid output rose, and at the same time there was an increased output of salt. Some patients got rid of their edema in this fashion in a few days, while others took several weeks. The excretion of albumin was high at first and tended to increase during the first days of improvement, after which it fell away to traces and eventually disappeared. The urinary sediment cleared up slowly. At first there were many granular casts, much blood and epithelium. Later hyaline casts appeared and remained for several weeks. A few blood corpuscles seemed to persist for months. The blood pressure fell to normal after suffi-

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cient rest in bed, and the dyspnea cleared up promptly. So far as could be discovered, there was nothing specific for trench nephritis in any of these features. They seemed typical of any form of acute nephritis with edema and hypertension.

Among complications, epileptiform convulsions were the most common. These developed in both mild and serious cases, with or without marked edema and with or without extreme degrees of hypertension. They were often associated with mild or maniacal delirium. They were not as alarming as they first appeared and were rarely fatal. Other complications were severe headache and gastro-intestinal indigestion, with nausea and vomiting or diarrhea. Pericarditis and pleuritis were almost never seen.

The eventual prognosis was good, as the mortality in a large series of cases was only 3 or 4 per thousand. The disease, however, did not clear up immediately, and every case was rightly considered as serious, and the patient unfit for any immediate duty.

The treatment for trench nephritis varied in different hospitals. As a routine in Base Hospital No. 5 in Boulogne, a rigid fluid intake and output chart was kept. For the first few days of observation the patient was given a fluid diet of 600 c.c. of milk and 900 c.c. of tea or water. As the fluid output rose, small amounts of bread and jam were added to the diet; and finally when the edema and dyspnea had disappeared, the patients were allowed eggs, porridge, small amounts of chicken, rice and potato, with such desserts as fruits or puddings. An effort was made to avoid diets containing much salt, protein or many calories. The most important single feature of such a course lay in the systematization. The same amount of fluid was given at the same hour every day. When food was added, it was divided carefully into three or four feedings, and each meal contained approximately the same amount of bulk and nourishment. On the whole,

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the patients did surprisingly well and were very comfortable.

Few drugs were used. Morphin or codein was useful for controlling cough, dyspnea or severe headache. Purgation was avoided as being liable to cause indigestion, and the bowels were regulated with enemas or mild vegetable cathartics. No patients were sweated. A few were given diuretics, but the results obtained were inconclusive. No effort was made to lower blood pressure, as it was difficult to obtain evidence that hypertension alone caused symptoms.

Bleeding seemed efficacious in stopping convulsions. In certain cases, 600 or 700 c.c. of blood were withdrawn, with immediate apparent relief. This procedure next to systematic dietetic regulation seemed most important.

The patients were sent to England on a hospital ship as soon as possible. They all traveled well and seemed to be unharmed by the trip.

The ultimate course in treatment consisted in managing the cases of trench nephritis as though they were cases of chronic nephritis. The diet was gradually made more liberal. After the edema and albuminuria disappeared the patients were allowed to get up, and were sent back to duty or were discharged from the army as unfit after a period of observation lasting for several months.

On the whole, the problem of nephritis in the soldier was essentially that of the treatment of trench nephritis. This was a type of acute nephritis of unknown cause and of remarkable frequency. It was characterized by its suddenness of onset, the symptom of dyspnea, the rapid occurrence and subsidence of edema, the commonness of uremic manifestations, and its low mortality. Clinical study of cases did not demonstrate anything specific as to the nature of the disease. Good results in immedite treatment were obtained by bleeding in the case of patients having convulsions, and by

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the systematic regulation of food and fluid intake in the less serious cases. The ultimate treatment was that of chronic nephritis.

The disease was serious from a military point of view, as it caused many casualties. However, even very sick patients stood a short journey well so that they could be sent promptly from the line to hospitals outside the zone of advance.

At present it appears that trench nephritis tends to heal and not to develop into a chronic progressive kidney disease. It will be necessary to follow cases for a much longer time than has been heretofore possible before this opinion can be established as fact.

## ABSTRACT OF DISCUSSION

DR. HAROLD FEIL, Cleveland: Approximately 100 of these cases have been casualty cases. There has been one striking point and that is the persistence of the hematuria and albuminuria without edema. These patients all resisted treatment in the hospitals for weeks and most of them have been discharged from the army for disability. All these cases were observed for a period of six months or more.

DR. E. P. JOSLIN, Boston: In the absence of Dr. Horace Gray and Major Vanderveer, I would say for them that each had a large series of similar cases under their supervision, and they have attempted to carry out what Dr. Fitz has suggested, i. e., to follow up the course of each case for several years. These gentlemen, who have worked at Vichy and Mesves, respectively, have collected the addresses of all these men, their records in the army, the names of their physicians, and hope to report on their condition later. As others have collected data of this description we should eventually have interesting facts relating to the after effects of this type of nephritis. In general, the innocuousness of the disease at the time of its height was obvious. Our patients at Mesves certainly did very well. The point was also obvious that these cases could be found if one looked for them. Following the influenza epidemic, a systematic examination of the urine of every man at the center was made, and then the cases promptly came to the fore—cases that would otherwise have been overlooked.

DR. H. E. JONES, Roanoke, Va.: I would like to ask the doctor a question: He did not give the type of the cases, except to state that chronic interstitial nephritis was absent. I would like to know what type of the disease he found among those cases of nephritis; that is, whether acute and

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chronic parenchymatous, or glomerular and tubular forms of the disease.

Dr. Homer F. Swift, New York: We have observed one interesting point in reference to this disease among American troops. From July to November, 1918, while the front line troops were living in trenches or "fox holes," exposed to cold and rain, very few cases of acute nephritis passed through the division triages. This winter, after our army arrived in Germany, although the troops were living under much better conditions we saw many patients suffering with acute nephritis similar to that described as trench nephritis. Living in trenches, therefore, does not seem to be a determining factor in the etiology of the disease.

Dr. Reginald Fitz, Boston: In answer to Dr. Jones, all I can say is that the clinical picture of acute trench nephritis was quite different and distinct from other types of nephritis. In addition there were cases of acute nephritis following tonsillitis, other infections or surgical conditions with marked albuminuria but without edema, hypertension or the dyspnea which was characteristic of the trench nephritis type.

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# THE TREATMENT OF CHEST INJURIES

MARION A. BLANKENHORN, M.D.
CLEVELAND

The treatment of penetrating wounds of the chest early in the war was an unsatisfactory venture offering little prospects of development, but at the end was a very satisfactory procedure promising much in the improvement of its technic and the enlargement of its scope.

Methods of examination of a wounded chest are precise; and in contrast to the abdomen or head, the damage done by a foreign body can be estimated and the treatment indicated. Foreign bodies entering the chest usually continue moving in straight lines; they produce discoverable lesions and can be located accurately by the roentgen ray. With proper appliances and training, the surgeon can invade the thorax to make anatomic repairs and remove infecting foreign substances. The effect of various infecting organisms in chest wall, pleura and lungs is still the greatest unknown quantity of the problem and the one that has yielded the least to our efforts.

It is with little satisfaction that I review the statistical reports of the various surgeons in France and England, and with even less satisfaction that I inspect my own records during two summers in forward area hospitals and one winter at the base. There were no invariable concomitants—warfare and its wounds, hospitals, surgeons and surgeons' ideas changed with kaleidoscopic rapidity. It would be inaccurate history to collect and attempt to coordinate the case records

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made by the various surgeons in France and England, and futile to recommend anything for the future.

The generalizations dealing with the pathology of penetrating wounds of the chest, and the principles of treatment here given, constitute the lesson learned from various British and American surgeons in handling approximately 500 cases.

Chest wounds in patients coming to forward area hospitals can be divided into five main groups on a basis of their gravity and the urgency of their surgical treatment: (1) open pneumothorax, the so-called sucking wound; (2) extensive parietal wounds involving ribs and muscles; (3) large retained foreign bodies; (4) infected hemothorax, and (5) simple hemothorax.

### OPEN PNEUMOTHORAX

It was apparent that there is no spontaneous cure for open pneumothorax. Anatomic repair is clearly indicated at once and is followed by almost miraculous, immediate relief. Patients who suck and blow a considerable volume of air through the wound were very uncomfortable, dyspneic and cyanotic, with a rapid, arrhythmic and irregular pulse of small volume. If untreated, they rapidly failed and died apparently from asphyxia. Simple stopping of the wound with a plug of gauze would revive a man in a few hours to the point at which he could undergo an extensive revision of his wound and closure. Patients who were so stopped up in advanced dressing stations arrived in good condition, frequently ready for immediate operation.

### PARIETAL WOUNDS

Parietal wounds, penetrating or nonpenetrating, demanded the same consideration that wounds did anywhere else, with the additional reason that indriven bone fragments became a source of infection. Broken ends of ribs projecting into the chest lacerate the lungs with each respiration, and occasionally do extensive

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damage to the heart and pericardium. Parietal wounds of less extent are a continuous source of infection to an adjacent hemothorax. A wound that connects with the pleural space by ever so small a sinus cannot be drained externally. As long as the aspirating effect of respirations is operating, the drainage is inward as well as outward. Such wounds, therefore, must be revised and completely closed. It was found preferable to convert a parietal wound into an open pneumothorax if necessary to make a thorough revision and a good closure.

Opening the chest freely has been found to be an easy and safe surgical procedure, well tolerated by a wounded man and causing very little shock. A very necessary part of the equipment needed to do surgery of the chest is a good nitrous oxid machine and a good anesthetist. When gas is given with a close fitting face mask and made to flow at considerable pressure, the lungs do not collapse when the chest is opened, and respirations and pulse are undisturbed. Furthermore, if anatomic repair of the chest wall and lung can be accomplished, the lung continues to function normally after operation.

### RETAINED FOREIGN BODIES

Retained foreign bodies are dangerous as a source of infection and of mechanical erosion of large vessels. Bullets and shrapnel balls that have not been deformed by impact with the soldier's equipment or bones, especially bullets, are surprisingly harmless. Shell fragments are invariably wrapped about with bits of clothing, and if they have one diameter as great as an inch, were almost never tolerated. To prevent empyema or lung abscess, they must be removed as soon as possible. Usually a man with a large retained foreign body is unfit for immediate operation on account of hemorrhage or shock or both. If considerable hemorrhage persists, he probably will never be fit to operate;

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resuscitation methods were unavailing, and it was not possible to operate for hemorrhage unless the hemorrhage was from a parietal vessel. Transfusion or infusion was distinctly detrimental if there was present a large hemothorax; for it could be clearly seen that the hemothorax was rapidly made larger, and the patients died suddenly with angina. The volume of fluid free in the chest apparently crowded the heart and lungs out of function.

Rest in a warm bed with plenty of fluids and morphin, usually restored these men to a condition that would tolerate an extensive operation. Removal of the foreign body was frequently an extensive operation. It required rib resection long enough to admit the hand into the chest. The fourth or fifth rib in the axillary space was best. The entire lung and chest could then be explored. The lung could be delivered down to the resection wound, and the foreign body with its pieces of clothing removed. If it was removed through healthy pleura, the pleura had to be sutured. If the foreign body was removed through a field of carnified lung, caused by the wound, it was usually unnecessary to make any closure or hemostasis, for this was quite well established by the process of carnification. How much should and can be done to carnified lung is a much disputed point. To me it was apparent that after gross contamination and necrotic tissue were removed, the less done the better. It was surprising how well such operations were tolerated, especially when done with gas given under pressure. Even the process of pulling on the lung caused very little change in blood pressure, pulse or respirations. After extensive lung injury and lung surgery, it was frequently impossible to maintain such a lung distended by the gas machine during operation or after closure; but gas pressure did prevent collapse of the sound lung during the operation, and prevented any disturbance of the heart and mediastinal vessels. Also

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it was not essential to a patient's well-being to have the wounded lung fully expanded after operation. In fact, it was thought by many that the lung should be kept collapsed and at rest after injury. But to accomplish this, collections of air or fluid in the pleural space was necessary, which introduced a new difficulty. One ingenious American surgeon accomplished partial collapse and rest by paralyzing the diaphragm with an injection of procain in the phrenic trunk, which was a rational and satisfactory procedure. Accumulations of blood were removed by pumping off the fluid and picking or wiping out all clot. The original wound of entry was revised, and when possible both wounds were closed tightly.

Infection of the pleural sinus was the greatest foe to the man who survived his anatomic injury. It was a surprise to all who observed it how able the pleural membranes are to combat infection. In all cases, whether operation was or was not performed, the degree of contamination must have been very great, for gross dirt could be seen and removed; but these very chests could frequently be closed and occasionally remain closed with no other help than aspiration. Pleural membranes combat infection best when in their natural position and condition. For this reason, it is essential to prevent accumulations of air or fluid.

After operation and closure, serous exudates appeared which had to be tapped. Sometimes aspiration, two or three times repeated, finished the course of treatment. In others, the fluid continued to reaccumulate and finally became purulent; but by this time the lung had become adherent to the chest wall in its upper portions, and we then had a small empyema cavity localized at the base of the chest. Stitches were removed from the resection wound, and drainage was easily accomplished. Introducing air or antiseptic solutions into the pleura to keep the lung at rest puts the pleural membranes at a disadvantage, and infection is invariably more severe.

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### HEMOTHORAX

The remaining two groups, infected and uninfected hemothorax, were at first indistinguishable and were managed alike. The degree of contamination and the amount of free fluid are the principal determining factors. The bacteriology of these infections, like the bacteriology of other wounds, is unsatisfactory and incomplete, providing help for some cases but rules for none. Each case must be judged individually and then chiefly on clinical evidence, temperature and pulse being of the greatest help to determine when a hemothorax is infected and drainage is necessary. Streptococci and gas-forming organisms, when found in specimen fluids, invariably foretold or indicated drainage; but even these organisms were known to have disappeared from a pleural cavity after one or two tappings.

It was a practice in forward areas to hold all uncomplicated hemothorax cases when possible for observation, and after three days if there was only moderate fever and specimens showed no growth to evacuate the patient down to the base. It was observed that infections sometimes lay dormant for much longer periods than three days. Presumably the blood retarded the growth of organisms, or colonies were included in the clot and had to digest their way out into the fluid. The natural culture medium in such condition is the hemorrhagic fluid, and the resistance to infection is at the pleural membrane. It was best, therefore, to reduce the medium and increase the resistance relatively by tapping off fluid and keeping it tapped off. It was seldom that hemorrhage was lighted up by tapping, and it was quite certain that empyema was prevented and sometimes cured by tapping. Occasionally, clot interfered with tapping, and the convalescence of such an individual was much prolonged. Usually almost the entire volume could be drawn off as a fluid which

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would not clot. In every case in which I have seen operation performed, clot either of whole blood or of almost pure fibrin has been found, so that it is my opinion that clot forms in all cases, but that defibrination is accomplished occasionally by some means.

Many uninfected hemothorax patients made rapid and complete recovery without any interference whatsoever.

Infected hemothorax must as a rule be drained freely; but drainage which causes complete collapse of a lung with the formation of a large cavity is very undesirable, for the collapsed lung frequently remains collapsed and the patient dies from a prolonged pyogenic infection of a large area of pleural membrane.

If free drainage can be delayed by repeated tappings until the partly expanded lung has established adhesions to the chest wall and only a small cavity is formed, the results are uniformly good. But there are certain cases, mostly when streptococci and gas-forming organisms are present, that become very septic and require drainage at once. Here a closed system of drainage by air-tight tubes and suction expands the lung, and provides the necessary adhesions to obliterate the empyema cavity. Irrigations of empyema cavities were generally practiced and found to be of considerable value—mostly to remove mechanically the accumulations of pus and clotted blood, and to diminish the number of organisms present.

Patients with free drainage usually did very well and could be evacuated to the base.

By the time patients arrived at the base in France, they had progressed to a stage that determined them either as having or not having empyema, provided medical lines of communication were normal. Frequently they were abnormal, and then all preconceived plans of management had to be revised, usually much to the detriment of patients.

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For cases without drainage, the management at the base was much the same as in forward areas.

For cases with drainage, the management was much the same as practiced in civil surgery, except that in some cases empyema cavities were treated by the Carrel-Dakin method and closed very promptly, frequently with very good results.

I had little opportunity to see patients treated by this plan, for it was done chiefly by the French or in England.

## ABSTRACT OF DISCUSSION

DR. ERNEST ZUEBLIN, Cincinnati: I am very much interested in Dr. Blankenhorn's paper, particularly from the standpoint of ordinary civil medicine. I would like to ask the doctor about the frequency of bilateral hemothorax, pneumothorax or hydrothorax. In a recent communication published in THE JOURNAL of the American Medical Association. Captain Green makes the statement that contrary to leading conceptions the simultaneous tapping of both pleural cavities is attended by hardly any harmful consequences. Of course, this is very important, and I would like to know whether Dr. Blankenhorn's experience along this line confirms such a view. Also of practical interest would be the recommended irrigation of the pleural cavity in case of emypema. I am curious to know what solution military surgeons advocate, particularly if two cannulas are introduced, one above and one below, in order to provide for a proper elimination and a clearing up of the purulent fluid.

DR. MARION A. BLANKENHORN, Cleveland: In reply to Dr. Zueblin's question about double pneumothorax, I have seen quite a few cases of double pneumothorax, but, of course, not double open pneumothorax. Closed pneumothorax was almost always the rule in chest wounds. What I might say about double hemothorax would explain double pneumothorax. Bullets frequently traversed both chest cavities, producing double pneumothorax and large or smaller double hemothorax, and if infection did not follow, the results were quite good. As to irrigations: We were using irrigations principally for mechanical reasons, and we used the surgical solution of chlorinated soda mainly. This solution dissolves fibrin from the pleural membrane and dissolves fibrin away from the opening of the tube into the wound, and we thought we were able to get rid of the original hemothorax more quickly by that process. Also, if organisms of putrefaction occurred, the odor was very promptly diminished, and we were also sure that the number of organisms was kept down. The plan of the management followed was to lay tubes along

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the walls of empyema cavities and give them routine irrigations, and when the bacterial count fell to a certain figure, close those cavities. I have not seen the statistics of those methods. Others have reported that if they did close in the presence of impending empyema, they would have to reopen, but if closure restored the normal condition to allow absorption of air and expansion of the lung, then they would have a smaller empyema. Frequently they would close and reopen a number of times. They considered this a much better method than to make no effort to expand the collapsed lung. The whole treatment was designed to obliterate the pleural sinus.

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# THE POSSIBILITIES OF PHYSICAL DEVELOPMENT IN CASES OF EFFORT SYNDROME BY MEANS OF GRADED EXERCISES *

BERTNARD SMITH, M.D.
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Patients who show the "effort syndrome" symptom complex often have a lessened muscular strength in addition to the neurosis that is generally present. This weakness may be limited to a few muscle groups, but it is usually general and may be very marked. This was true with the men who entered this hospital from the American camps during the summer of 1918 and, because of this picture of poor muscular development, graded exercises were among the first methods adopted for an intensive study of the effort syndrome condition. Lewis and his co-workers[1] have used grouped exercises, requiring varying degrees of effort, in estimating the future military usefulness of these men, and, from the results, those men unfit for military service were listed earlier for discharge and the individuals retained in service were classified for duty by the grade of graduate work they could successfully carry through.

In the studies at Lakewood all effort syndrome cases on the cardiovascular wards were admitted to the exercise classes, even though the severity and persistence of the symptoms indicated a definite unfitness for any military service. In trying various forms of exercises, it was found that the confidence of the men in their ability to carry on must be secured and maintained before any efforts toward improved strength could be successful. Sharp, abrupt commands were destructive to this confidence and caused a return of overwhelming symptoms as easily as too severe work. After considerable experiment in arranging

* Report from studies made on the Cardiovascular Service, U. S. Army General Hospital No. 9, Lakewood, N. J.

1. Report of Medical Research Committee, Series No. 8, 1917.

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exercises for so general a reconstructive problem, the "unit system" was adopted.[2] The eight "units" include all muscle groups and their order is the same in each exercise grade. This report is concerned with the results from these exercises in the attempt to overcome the existing physical weakness.

An estimation of the total strength of each patient was made when he entered the hospital, and again when he was ready for discharge or had completed the highest exercise grade. For these estimations the spring balance resistance test of Dr. E. G. Martin was used, the technic of which is briefly described at the end of this report. Figures for the strength of normal men in active military service were obtained from tests made on one hundred men at Camp Dix, N. J. These men had had a period of full, hard training covering from ten to fourteen months, which they had completed without symptoms. The range and average of these normal estimations are given in Table 1. It was evident from the normal figures obtained that the weight is an important factor if a comparison of strengths is to be made. Consequently, all tests have been computed for total strength (Str.) and for the ratio of strength to weight (Str./Wt.).

TABLE 1.—Strength Estimations on 100 Normal Men in Active Military Service*

| Range | | | | Average | | | |
|---|---|---|---|---|---|---|---|
| Strength | Weight | Str./Wt. | Age | Strength | Weight | Str./Wt. | Age |
| 1390–2830 | 54.6–88.5 | 23.2–30.8 | 19–40 | 1655 | 67.6 | 25.6 | 24.1 |

* All figures for strength and weight are in kilograms.

The clinical history of cases of effort syndrome among soldiers may show that the condition was present previous to entrance into military service. Others give equally definite evidence that the symptoms have developed during the period of active duty. It will be convenient to arrange the cases here reported under these two groups, according to the onset and duration of the general symptoms.

### I. CASES WITH SYMPTOMS BEFORE MILITARY SERVICE

Of the 300 effort syndrome cases here reported, 56 per cent. give a history of symptoms having been experienced for a number of years

2. Smith, Bertnard: J. A. M. A. **72**:103 (Jan. 11) 1919.

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before entrance into the army. The majority of these men have performed very little, if any, real active duty. They have found their way into a hospital or infirmary soon after induction into service.

1. *Type of Intelligence Defect.*—Among the men who give a history of this long duration of symptoms, thirty-five showed definite but varying degrees of defective intelligence.[3] These give differentiating characteristics that deserve attention. They are lacking in both physical and mental drive, and can be neither persuaded nor forced to do their full part in training. They have a persistently poor coordination, and go through the work of the various exercise grades with joints sagging and muscles relaxed. These men are without ambition and show a stubborn resistance to any effort that is made to change them from the low economic level to which they have become adapted. Their ability to adjust themselves to a new environment, especially to military conditions, is slow and limited. The physical weakness may be extreme (Table 2), but this is not constant, and the less severe cases may have a strength that is very near normal.

TABLE 2.—COMPARATIVE STRENGTHS OF CASES OF EFFORT SYNDROME BEFORE AND AFTER EXERCISE

| | No. | On Entrance to Ward | | | On Discharge from Ward | | |
|---|---|---|---|---|---|---|---|
| | | Range of Str. | Range of Str./Wt. | Average Str./Wt. | Range of Str. | Range of Str./Wt. | Average Str./Wt. |
| Symptoms of long duration: | | | | | | | |
| 1. Intelligence defect | 35 | 175-1870 | 3.9-24.4 | 15.1 | 780-2096 | 10.7-28.0 | 21.5 |
| 2. Physical invalidism | 110 | 344-1475 | 6.7-22.8 | 14.8 | 883-2061 | 15.3-36.3 | 24.8 |
| 3. Unclassified | 23 | 492-1619 | 10.8-34.0 | 21.0 | 1124-1420 | 18.9-36.0 | 26.3 |
| Symptoms recent: | | | | | | | |
| 1. After strain | 70 | 600-1981 | 9.0-26.0 | 20.4 | 1225-2180 | 22.2-37.6 | 26.9 |
| 2. After infections* | 62 | 593-1621 | 9.7-28.2 | 19.3 | 1249-1944 | 19.3-31.9 | 25.8 |
| After rheumatic fever† | 33 | 822-1706 | 15.3-27.4 | 21.3 | 966-2105 | 18.6-29.8 | 25.4 |

* Not including cases following rheumatic fever.
† Not included in cases of effort syndrome.

Satisfactory strength estimations are often difficult to obtain in severe cases of the defective intelligence type because of the lack of cooperation. Consequently, it is doubtful if the low figures represent true strength values. In Chart 1 curves are given which show variations in strength estimations made at three periods of the same day,

3. Campbell, C. Macfie: J. A. M. A. **71**:1621 (Nov. 16) 1918.

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both at the time of entrance into the hospital and at the time of discharge. The ten curves are from estimations made on men of the defective intelligence type who had low strength readings. At the time the first observations were made the day was spent quietly on the wards or with the various routine examinations. The second group of curves are from tests made after two or more months had been spent in daily exercise classes and when the day was well occupied with educational or vocational work, together with the classes in physical training. There is a general gain in strength, but the type of curve persists. Curves obtained in the same way from the normals and from other types of effort syndrome cases are of no constant form. Ryan tests[4] on this type of constitutional inferiority show an early fatigue. While these ndividuals can advance through the exercise grades with but few symptoms and with some slight gain in general strength, the fact that they will always put the least amount of effort possible into the work is probably explanatory of the early, persistent fatigue. With the gain in general strength there is a betterment of the general health, but the mental sluggishness remains unchanged and the men will not make any independent effort to avoid slipping back into the former condition.

2. *Type of Chronic Physical Invalidism.*—The majority of the men whose symptoms have persisted for a long time give a picture that is in rather sharp contrast to the type just considered. Here the physical weakness forms the predominant and constant picture. These men have "never been strong," or trace the beginning of the trouble to an illness in early life, frequently in infancy or childhood. A diagnosis of serious heart trouble, combined with the watchfulness of oversolicitous parents, form important contributing factors. The patients have learned to interpret the symptoms that appear after physical exertion as warnings of serious overstrain.

Since the fear of the results of this overstrain has become very magnified, it is important that physical training for these men should begin with exercises of so mild a form that no symptoms result. Advancement in the graded work must be slow and so well graduated, especially in the lower classes, that the individual is not too conscious

4. King, John T., Jr.: Arch. Int. Med. **23**:527 (April) 1919.

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of the increased effort demanded. Some symptoms may be experienced on beginning a higher grade which will disappear after two or three days of perseverance. Men of this type usually have a normal men-

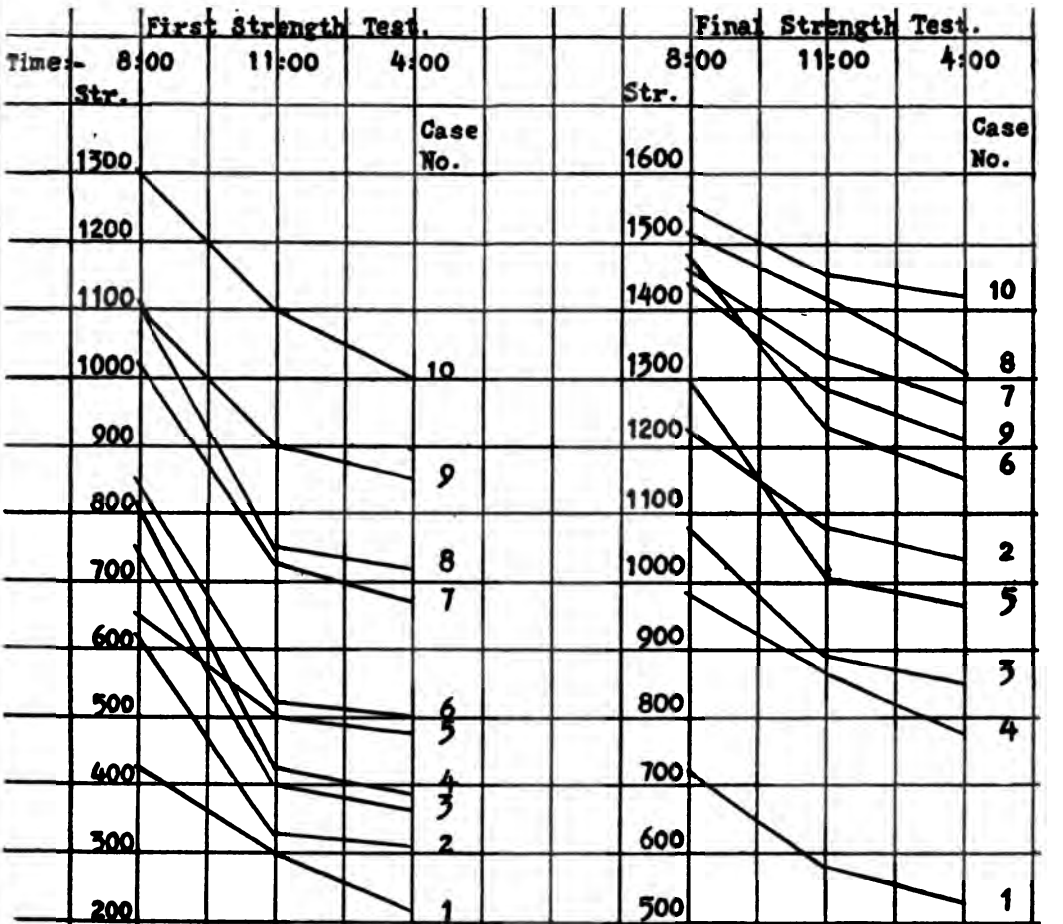


Chart 1.—Fatigue curves in severe cases of effort syndrome. Defective intelligence type. Curves from estimations of strength made before and after work in graded exercises.

tality, and such cases can be found among college and university students. Some manifestations of neurosis are usually seen in these men, but this factor is usually subordinate to the physical weakness.

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However, progress in the exercises may depend more on the ease with which self-confidence can be developed and maintained than on the general strength present at the beginning of the work. The man with the lowest strength of 334 kg. (Table 2) returned to duty at the end of two months, having completed all the exercise grades with a final strength of 1,658 kg. These men differ from the cases with defective intelligence, in that their cooperation is complete as soon as confidence in their own ability to carry on is restored. After they are able to take part in the more vigorous games and exercises, future progress is usually secure. The pride in their increased strength and their joy in losing the stigmata of being a weakling are sufficient to carry them ahead and should prevent them from falling back into old habits.

TABLE 3.—Time in Exercises of Effort Syndrome Types, with Final Grade Reached

| Type* | | No. | Weeks in Exercises | | Final Grade at Time of Discharge, with Average Str./Wt. | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Grade I | | Grade II | | Grade III | | Grade IV | | Grade V | | Grade VI | |
| | | | Range | Average | No. | Str./Wt. | No. | Str./Wt. | No. | Str./Wt. | No. | Str./Wt. | No. | Str./Wt. | No. | Str./Wt. |
| III | 1 | 35 | 10-19 | 12.5 | 1 | 10.7 | 2 | 14.6 | 2 | 15.4 | 6 | 17.2 | 9 | 22.4 | 15 | 25.3 |
| | 2 | 110 | 9-16 | 10.6 | 0 | .... | 0 | .... | 3 | 16.2 | 15 | 18.6 | 42 | 24.1 | 50 | 26.4 |
| | 3 | 23 | 8-13 | 10.5 | 0 | .... | 0 | .... | 0 | .... | 4 | 19.3 | 10 | 24.4 | 9 | 25.8 |
| II | 1 | 70 | 5-13 | 8.4 | 0 | .... | 0 | .... | 0 | .... | 0 | .... | 5 | 24.8 | 65 | 25.5 |
| | 2 | 62 | 8-12 | 9.3 | 0 | .... | 0 | .... | 1 | 19.4 | 5 | 21.1 | 14 | 24.8 | 42 | 25.6 |
| After R. F. | | 33 | 7-13 | 8.6 | 0 | .... | 0 | .... | 0 | .... | 4 | 19.8 | 16 | 24.1 | 13 | 25.2 |

* Group and type numbers correspond to those in Table 2.

In the cases here reported, 110, or 37 per cent., belong to this type of physical inferiority. Of these, 84 per cent. advanced into the fifth grade of exercises in an average time of 10.6 weeks, and took part in active athletic games. The figures in Table 3 that represent the men who only reached the third and fourth grades should not be interpreted as meaning a permanent limit for these individuals. These grades were their limit only for the time they were in the hospital. Proper training after discharge should secure further improvement.

A special exercise class was formed with twenty-five patients selected from the group where symptoms were of long duration, with

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the desire to test the results of a more intensive training under close supervision. These men began with various forms of track work and athletic games, in addition to heavier exercises than those in the regular work for lower grades. The results with fifteen men of the chronic invalidism type are given in Table 4. The average ratio of strength to weight (Str./Wt.) on beginning work in this special class was 15.0, and, after an average of ten weeks of training, this ratio had increased to 23.6. With the 110 men who had their training in the general classes, the first strength to weight ratio averages 14.8. After an average of 10.6 weeks of work this ratio average had increased to 24.8. In the more intensive work it was definitely more difficult for the men to maintain the degree of effort required without becoming too conscious of the general effort syndrome symptoms.

TABLE 4.—RESULTS OF INTENSIVE TRAINING IN CASES OF CHRONIC INVALIDISM TYPE

| Name | First Str. | First Str-/Wt. | Weeks in Class | Final Str. | Final Str./Wt. |
|---|---|---|---|---|---|
| B. F. | 1006 | 16.7 | 9 | 1240 | 20.6 |
| F. W. | 1083 | 18.8 | 9 | 1250 | 22.7 |
| G. H. D. | 757 | 12.8 | 12 | 1450 | 24.6 |
| G. L. | 1499 | 21.0 | 11 | 1881 | 25.7 |
| E. M. | 1141 | 18.3 | 8 | 1628 | 25.2 |
| E. J. G. | 689 | 12.5 | 12 | 1215 | 22.0 |
| H. R. | 972 | 14.8 | 9 | 1588 | 25.6 |
| G. H. | 830 | 13.8 | 8 | 1678 | 26.9 |
| C. H. | 1175 | 18.2 | 10 | 1445 | 23.9 |
| F. P. | 441 | 8.0 | 12 | 1022 | 18.5 |
| B. D. | 830 | 18.5 | 19 | 1220 | 28.5 |
| O. S. | 842 | 14.3 | 10 | 1365 | 23.1 |
| S. G. | 791 | 12.7 | 10 | 1334 | 21.6 |
| W. H. | 1335 | 20.4 | 13 | 1820 | 27.7 |
| W. E. H. | 218 | 4.3 | 9 | 1332 | 22.6 |
| Average | 903.7 | 15.0 | 9.9 | 1431.7 | 23.6 |

3. *Unclassified Type.*—In Tables 2 and 3 cases are included under "unclassified type" that had predominating nervous symptoms. Some degree of neurosis is present in all effort syndrome cases and must be recognized in a successful treatment of this condition. But there were a few men whose progress in graded work was so controlled by the nervous factor that they deserve a separate grouping. Frequent fainting attacks, complaint of severe dizziness, independent of exertion and other indications of a marked nervous instability separate these men from the other types and limit their advancement in the exercises. The general strength shows a wide variation, and there is usually no indica-

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tion of a defective intelligence. These men require neuropsychiatric attention in addition to the physical training.

### II. CASES DEVELOPING SYMPTOMS DURING MILITARY SERVICE

As the period of active fighting for our troops continued, the type of cases admitted to the wards with the official diagnosis of cardiac disorder functional changed from the group with long continued symptoms to men with a greater strength in whom the condition had developed during active army duty. These patients have a previous history that is negative for the usual effort syndrome symptoms. The symptoms they present on entrance to the hospital may differ in no way from those of the more chronic group.

1. *After Nervous and Physical Strain.* — Of the 132 men who belong to this group with a recent onset of symptoms, seventy, or 53 per cent., give no etiology other than the severe nervous and physical strain of military duty. Many of these men were engaged in rather sedentary occupations in civil life, but had always considered themselves well and strong. They went through the period of training in the American camps without difficulty, and only eight of the seventy broke down during the intensive training overseas before being sent to the front. The first strength tests on these men give higher average values than are found in the cases with long continued symptoms. Also the confidence of these men in their ability to get back into condition is not so thoroughly destroyed. Many are able to advance rapidly through the exercise grades and are free from symptoms after four to six weeks of graded work. Of the seventy men, sixty-five completed all the grades of exercises without symptoms in an average time of 8.4 weeks. These men are a much milder type than are those reported by the British workers. It must be remembered that the American troops were in active fighting for a much shorter time.

2. *After Infectious Disease.*—A group of sixty-two men were admitted to the cardiovascular service with a diagnosis of a functional cardiac disorder following some infectious disease. The cardiac symptoms had developed during the convalescence or after the patient had returned to duty. Some of these men were admitted from American camps and were in an earlier stage of convalescence than were the

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men returned from overseas. This may account, in part, for the lower average strength on admission than was found in the case of the men who broke down under strain. The gain in strength and the disappearance of the symptoms in graded exercises is about equally rapid in these two types. However, some complication may persist in the postinfection cases which limits their advancement in the graded work.

Patients giving a history of symptoms developing after acute rheumatic fever have not been included in this report of effort syndrome cases. The common incidence of rheumatic fever in organic heart disease influenced the separation of these cases into an independent group, even though a thorough study and close observation during a long period of graduated work failed to reveal any organic defect. In Tables 2 and 3 are appended the observations in thirty-three cases in which the men developed symptoms after acute rheumatic fever. It is shown that 88 per cent. of these men advanced into Grade 5 exercises, and 39.5 per cent. completed the highest (6) grade. In cases of the general postinfectious type, 90 per cent. of the men completed Grade 5, and 68 per cent. finished the work of all grades successfully. Men with arthritic symptoms are not included in this postrheumatic number, but among the postinfectious patients, a number had persisting complications. Arrhythmias due to some myocardial defect are more frequently found in the postrheumatic cases during the exercise work, and these men must be advanced in the grades only under the most careful observation.

## GENERAL DISCUSSION

The classification used in this report is purposely broad, since the desire is to emphasize the fact that variations in type can occur among cases presenting the effort syndrome symptom complex rather than to advocate any fixed terminology. It is believed that a more rational understanding of this condition can be reached and a more successful treatment carried out when an appreciation of the variations in type is recognized and an individual study of each patient is made.

Men who have presented symptoms for a long period of time, and who have failed to stand up to their earlier military training, cannot be built up to a normal strength in the short time and by the intensive

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methods necessary during a military emergency. Such treatment will only increase the severity of the symptoms, and the patient is given a more fixed idea of invalidism in consequence. The treatment is more properly a civil problem, and should receive its deserved attention during the time of peace. The importance of recognizing this is emphasized when we consider the number of recruits who were found to be unfit for military service because of a poor physical development unassociated with any organic disease.

Men belonging to the type of chronic physical invalidism have a military value if they can be developed to a condition of greater strength and, what is even more important, they can be made fit to successfully undertake more active work in civil life with an improvement in their economic life. There is an increase in their military value that is, to some degree, in direct proportion to their ability to stand up to more active civil work.

Men showing definite intelligence defect present the most discouraging problem in treatment. The mental sluggishness does not permit of a satisfactory cooperation from these men.

Where the symptoms have been of short duration and acquired during active military duty, the general strength shows a higher average and the men lose their general symptoms more rapidly. This is especially true where the condition follows the physical and nervous strain of front line fighting, and the symptoms are no more persistent than in the cases here reported. If comparisons are to be made, it must be remembered that our troops were not in the fighting line so long as were the allied forces. Also, the signing of the armistice removed any fear of a return to active duty which might have been an important factor in delaying recovery.

Men who develop the effort syndrome after acute infections will improve rapidly in strength and in symptoms through graded exercises. They may be limited in their advancement through the grades by some resulting complication. When carried out under proper supervision, well graded exercises should form an important part in the late treatment of infectious diseases.

No attempt has been made in this report to give the percentages of men returned to duty. It is doubtful if any reached the front line in time to take part in active fighting for a sufficient length of time to be

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a satisfactory test. A number have been heard from through personal communications, several months after they had returned to civil life, and these report having taken up more active work than had been their former occupations and having been free from symptoms.

All strength estimations included in this report were made with the assistance of Dr. John T. King, Jr., to whom I am indebted for most efficient cooperation. I wish to express my appreciation of the work done by the men who directed the classes in graded exercises: G. J. Altman, Paul M. Krimmel and Morris Kaufman. The general plan adopted for the exercises was worked out by these men and the success of the work was largely due to their able and faithful services.

## THE MARTIN STRENGTH TEST

### SPECIFICATIONS

*First.*—A self-registering spring balance and ice scale. A very satisfactory scale is now furnished by John Chatillon and Sons, 85 to 93 Cliff Street, New York City, as "Special Strength Tester. Style No. 100 B." The capacity should be designated in ordering and can be had in pounds (200) or kilograms (100).

*Second.*—An upright post with a hand hold. A stout post, 4 inches square and 6½ feet high, mounted on a firm base. The post should be smooth and have the corners rounded off. A strong rod, 1½ inches in diameter and 24 inches from the post, should be placed as a hand hold.

*Third.*—A stout table of ordinary height, not less than 6½ feet long and 2½ feet wide, with a cleat of wood about 2 feet by 1½ inches by 3 inches, securely fastened on the top of the table across one end, with the greater diameter vertical.

### PROCEDURE

Ten groups of muscles are tested: 1. Right pectoral; 2. left pectoral; 3. right wrist flexors; 4. left wrist flexors; 5. right forearm flexors; 6. left forearm flexors; 7. right thigh adductors; 8. left thigh adductors; 9. right thigh abductors; 10. left thigh abductors.

After the loop of the balance is adjusted and the subject is in position, the command "hold back" is given. At this command the subject contracts the muscles of the group being tested with all his power and, simultaneously, the operator pulls on the spring balance. The tension must be developed as rapidly as possible, without jerking, and must be increased until the resistance of the subject is entirely overcome.

### TECHNIC

Pectoral Groups: The subject stands at attention, with the middle of his back pressed firmly against the upright post, and the hand of the arm not being tested grasping the hand hold. The adjuster stands directly in front of the subject, facing him and places the loop of the balance about the arm to be tested, just above the elbow. With one hand he holds the loop in position and, grasping lightly the hand of the arm to be tested, draws the arm across in front of the subject's body as far as possible, keeping the arm straight

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and as close to the body as can be done and still give clearance for the loop. At the command "hold back," the subject makes all the resistance possible and the operator develops sufficient tension to draw the arm down to the side of the body. The pull must be discontinued before the arm has been drawn beyond the vertical line.

Wrist Flexors: The subject stands beside the upright post with the arm to be tested so flexed as to bring the forearm horizontal. The back of the forearm must rest against the post, and the hand project beyond the post to the ulnar process. The adjuster stands directly in front of the subject's palm. With one hand he holds the subject's wrist against the post and, with the other, he holds the loop in place. The loop is placed so that its middle is directly over the crease at the base of the fingers. Keeping the fingers straight, the subject's hand is flexed at the wrist. The operator pulls at an angle just less than 90 degrees from the plane of the subject's hand. Tension must stop as soon as the hand begins to yield.

Forearm Flexors: The subject lies on his back on the table, with his heels pressed firmly against the cleat. The adjuster stands at the subject's left for both flexors. His right hand holds the subject's forearm in a position of flexion, about 15 degrees toward the shoulder from the vertical, and places the loop about the wrist so that its upper edge is at the crease in the skin at the base of the hand. The operator stands at the foot of the table. Tension is relieved when the forearm reaches the vertical.

Thigh Adductors: The position of the subject is the same as for the forearm flexors, except that he presses against the cleat with only the foot of the leg that is not being tested. The adjuster stands at the foot of the table. With one hand he places the loop in the hollow just above the malleolus, seizes the subject's heel with the other hand, lifts the leg until the heel is just high enough to clear the other toe, and then draws the leg into extreme adduction. The toe of the leg to be tested must be upright. The operator stands at the side of the table, develops tension at the word of command and draws the leg outward from the side. Tension is relieved as soon as the leg has been drawn into line with the axis of the body.

Thigh Abductors: The positions of the subject and adjuster are the same as in the last test. The loop is adjusted as for the adductors but the pull is in the opposite direction. The leg is drawn out beyond the midline of the body to an angle of about 15 degrees, and the effort of the subject is to prevent the operator from drawing the leg into line with the body. The tension is relieved when the leg reaches the midline.

*Calculation.*—The sum of the strength shown by these ten groups of muscles constitutes in men 17.7 per cent. of the entire strength as found by this system of testing. To calculate the total strength therefor, the sum of these ten determinations must be multiplied by the reciprocal of 0.177, which is 5.65. The product thus obtained is the figure for the strength of the individual.

NOTE

This technic is printed with the consent of Dr. Ernest G. Martin, to whom I am indebted for permission to use the test. I wish also to express my appreciation of the suggestions regarding the technic that were given by Prof. F. S. Lee.

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## ABSTRACT OF DISCUSSION

DR. EVELETH W. BRIDGMAN, Baltimore: After eight months of active service in a base hospital in France I was sent to England to work under Dr. Thomas Lewis, the pioneer in effort syndrome work. Two months later I was recalled to France and assigned for duty at the convalescent section of a group of base hospitals, where I saw 5,000 cases of effort syndrome. The histories and follow-up reports serve as the basis of these remarks. In the absence of a cardiovascular test that would be comparable to the phtalein test for kidney function, Dr. Lewis had instituted graduated exercises which proved not only a satisfactory method for the classification of cases of effort syndrome, but also served as an excellent therapeutic procedure. A similar series of graduated exercises were adopted for these patients in the American army with excellent results. Nearly all the patients entering the camp had symptoms which make up the effort syndrome. Ninety-one per cent. were sent back to full duty after a stay determined by their ability to perform work comparable to that of active duty, an average of eighteen days. One and one-half per cent. were reclassified for light duty because of effort syndrone. This group was made up in equal proportions from patients with varying diagnoses on admission. That is, the effort syndrome cases were found just as frequently among patients who had had pneumonia or influenza, as among those who had been gassed or wounded. In other words, effort syndrome is not alone a wartime condition but is met with constantly in the consulting room of every physician. Not far from our camp was a hospital devoted entirely to the treatment of war psychoses. As a result of exchange visits, the physicians of both institutions learned to appreciate the similarity of the symptoms to be found in the two places. In many instances we agreed that the original diagnosis had depended in large part on the specialty of the doctor who had first seen the patient, the psychiatrist favoring the diagnosis of anxiety neurosis, the internist that of effort syndrome. In this way, at our camp, we came to appreciate that effort syndrome is not a diagnosis, it is a symptom complex that will generally allow a fundamental diagnosis. Just as jaundice or tachycardia may be present in different diseases, so effort syndrome may develop in numerous pathologic states, and we, therefore, came to make multiple diagnoses in these cases: anxiety neurosis, effort syndrome, constitutional inferiors effort syndrome, underdevelopment effort syndrome. Perhaps such a conception of multiple etiologic factors will afford the best type of treatment. We found that the base hospital, before the establishment of convalescent camps, had been allowing too short a period of convalescence before the return of the patient to full duty, and that, as a result, many soldiers, apparently

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cured, would later return with signs of effort syndrome. This return was almost entirely prevented by the prolongation of the period of convalescence, till the patient was actually able to do work comparable to front line duty. The average case of mumps necessitated thirty-nine days before complete recovery; tonsillitis, thirty-four days; herniotomy, fifty days; tonsillectomy, thirty-seven days. The need for so long a period of convalescence is probably not appreciated by most physicians. Of different etiologic factors, constitutional variation in individuals is important. Experience in the training of track athletes develops the opinion that there are two outspoken types of men, with numberless gradations in between, the one capable of supreme concentration of energy for a short period of time, the dash man, the other slow going and very resistant to fatigue. The training of the two groups is quite different. The training of the sensitive dash man is always difficult, and his work reflects his mental attitude where the worry of a failure in recitation will be recorded by a reduction of his speed; while the phlegmatic distance runner plods along comparatively uninfluenced by his surroundings. Reverse these men and the long distance man is a failure in the sprints, but the dash man will not only be unsuccessful in his new work, but he may develop typical signs of effort syndrome. In the army all men are subjected to a routine training. The distance runners carry on well, but the dash men often develop effort syndrome. Individuality is just as real a condition in the army as in civilian life. There are two important impressions that I carry back to civilian practice, first, that effort syndrome is not a disease entity but is made up of symptoms resulting from many different etiologic factors, and, secondly, that prophylaxis is important to consider, and that among helpful procedures to this end, a sufficient period of convalescence is important.

Dr. W. S. Thayer, Baltimore: This paper and the remarks that have been made touch on a subject which is likely to be one of the most important medical outcomes of the war, namely, the direction of the attention of the medical profession to the importance not only of the care of these subnormal individuals, but to the care of convalescents. In practice, the doctor, as a rule, has little opportunity to direct convalescence, and having little opportunity has subconsciously lost interest and lost a sense of responsibility in that which we can do for the individual. Here for the first time the convalescent has not been his own master; the government has demanded that he be hurried back to the service so soon as possible, that every measure be adopted for his early and complete rehabilitation. This has opened an important field. Bridgman's figures are of more than passing interest. The fact, for instance, that of a consider-

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able number of instances of acute tonsillitis in healthy young men, 20 to 30 years of age, the average under the best of conditions are able to pass the test justifying their return for duty only thirty-four days after the onset of the disease, gives us something to think about. When one reflects on the late complications of tonsillitis, of the endocarditis that often follows when we have fancied the patient well, one asks himself how much of this might be prevented by holding on to the patient and directing his convalescence properly. The realization of the importance of caring for our patients during convalescence, and of the desirability that institutions be started where convalescents may be cared for, is, I believe, going to be one of the striking outcomes of this war.

Dr. Bertnard Smith, Los Angeles: I have been much impressed with the importance of physical development in the work at Lakewood. Exercise tests are of great value in cardiac diagnosis and well graded work should be given a more important place in the treatment, especially in functional cardiac disorders. The care of effort syndrome cases now becomes a civil problem and I consider it necessary that our hospitals and outpatient clinics realize the need of physical development in the treatment of these cases. Exercise tests have shown us that the convalescence after severe infections is much longer than has been believed. This was found true at Lakewood, as Dr. Bridgman reported. Myocardial defects are often revealed, and the degree to which the patient's physical effort should be limited can be gaged more definitely. Effort syndrome cases with symptoms of long duration may show an early fatigue. Dr. John T. King has reported this in his work with the Ryan test and compares his results with the Martin strength test. This early fatigue may persist even through the higher exercise grades and is probably due to the lack of physical drive on the part of the individual. But fortunately most cases show an improvement in strength and symptoms that is accompanied with the loss of this early fatigue and these are the men who deserve attention.

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# TRENCH FEVER IN THE AMERICAN EXPEDITIONARY FORCES

HOMER F. SWIFT, M.D.
NEW YORK

Trench fever is a disease that has been recognized and differentiated from other morbid conditions during the recent war. While there is little doubt that it existed previously in some place or places, there is no doubt that to all medical officers it presented new problems in diagnosis, prognosis and treatment.

There are descriptions of the disease and reports of its occurrence in all armies on the western front. But from the mass of reports it is impossible to determine the original source of the infection or the line of spread from area to area. The constant movement of troops in an army, the ever changing composition of every unit, make epidemiologic studies very difficult and accurate reconstruction of the mode of diffusion of a well known disease virtually impossible. When we consider that on the western and eastern fronts and behind the lines there were troops from nearly every country in the world, it is easy to understand how an unknown disease, which might have been endemic in some out-of-the-way country, might rapidly gain a foothold among men who had not developed any natural immunity.

The difficulties in collecting accurate statistics concerning trench fever were increased by the recency of its discovery. It was never made a reportable disease because the conditions which must be fulfilled to make reporting compulsory could not be clearly defined. For many months most medical officers were ignorant of the existence of the disease, let alone its manifestations in the patients under their care. To the entire

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medical profession the nature of the disease was more or less a mystery. The fact that the term "pyrexia of undetermined origin" (P. U. O.) was applied in the diagnosis of most cases is sufficient indication of the general attitude toward the malady. Although the specific nature of the infection was established by McNee, Brunt and Renshaw in 1916, it continued to be confused with other acute infections. The term "pyrexia of undetermined origin" was applied to all these conditions, even though it usually implied trench fever.

### DIAGNOSIS

While it is fairly easy to describe the general disease trench fever, it is much more difficult to make an exact diagnosis in many individual cases. This difficulty arises from the variety of manifestation.

Fever, which is the most common of all the symptoms, does not always follow any one course. Various efforts have been made to separate the types of fever into two or three groups. The most comprehensive classification is that of the British Expeditionary Force Trench Fever Commission:

(*a*) Relapsing, either regular or irregular.

(*b*) One single short bout resembling influenza.

(*c*) Continuous fever extending over a longer period, resembling in its form typhoid or paratyphoid fever.

While such classification is useful, it should be emphasized that the cases do not always fall into one of these groups. A case with regular relapses may later have long continued low-grade fever. The febrile relapse may occupy only four to six hours, and hence may be missed if only morning and evening temperatures are taken. Again, the entire fever may continue less than forty-eight hours. The case with three or five days continuous fever without a subsequent relapse may be easily mistaken for influenza.

Charts 1 and 2 illustrate the different types of fever that may occur from inoculating different individuals

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with the same strain of virus. They are abstracted from charts of the volunteers inoculated by the American Red Cross Trench Fever Commission.

The other clinical manifestations of trench fever, taken separately, are even less diagnostic. The sudden

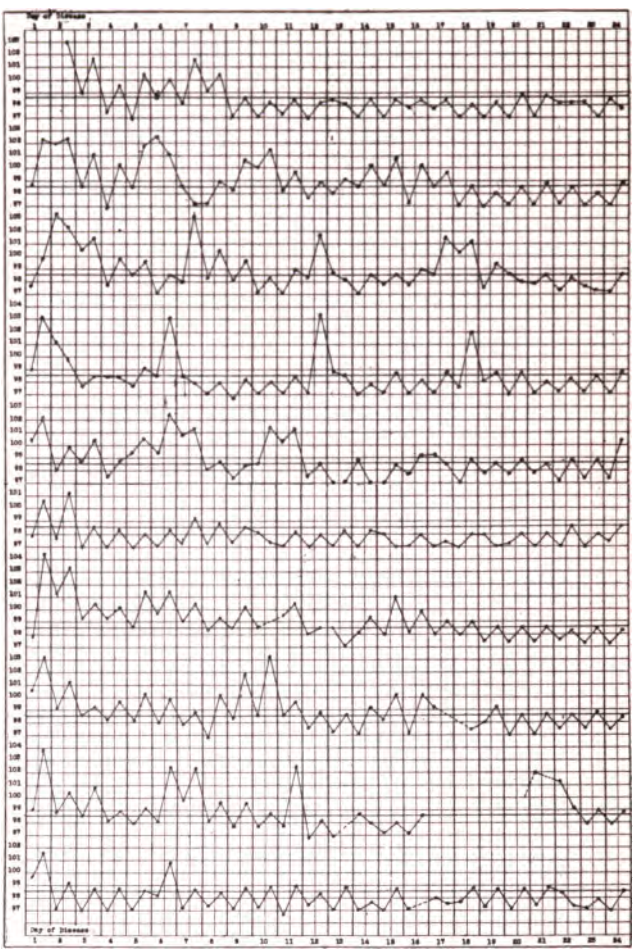

Chart 1.—Different types of fever that may occur from inoculation of different persons with the same strain of virus. In the next to the last case the temperature was normal from the twelfth to the fiftieth day. In this chart the temperature after the blank space marks the febrile relapse on the fifty-first and fifty-second days.

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onset with headache, general muscular pain and anorexia is seen in other acute infections. The "pink eye" resembles the conjunctival congestion so frequent in influenza. The nystagmus, elicited on extreme lateral rotation of the eyeballs, may be present normally and is seen in other febrile states.

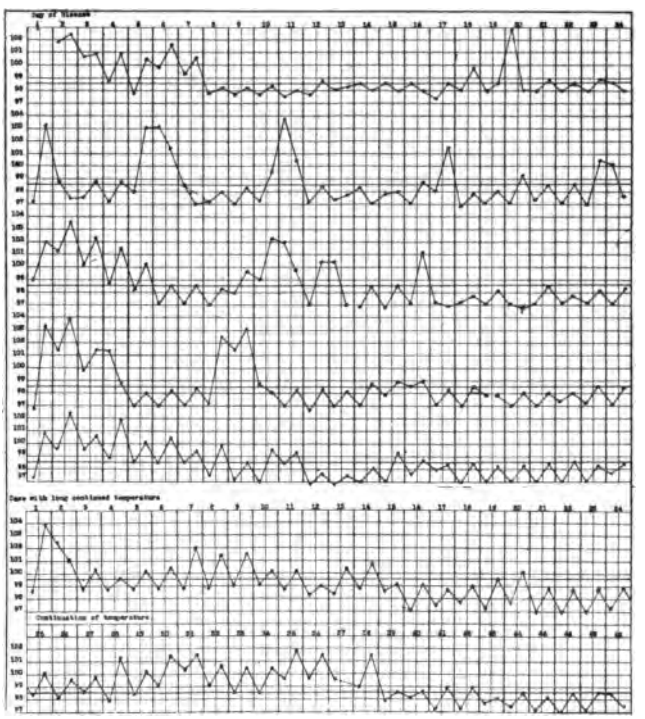

Chart 2.—Different types of fever that may occur from inoculation of different persons with the same strain of virus.

An enlarged spleen is found in typhoid fever and malaria, either of which may be mistaken for trench fever. The trench fever spleen, however, is harder than that found in typhoid fever and not so large as that often present in malaria.

The trench fever rash may be mistaken for typhoid rose spots, if the evolution of the individual macules

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and the relation of their appearance to the other manifestations of the disease are not carefully recorded.

The neck pains may suggest meningitis; the pain in the back, lumbago; and the abdominal pains, appendicitis or acute peritonitis. The joint and muscle pains are often mistaken for subacute or chronic rheumatism or myalgia. The painful joints in trench fever are not swollen. The shin pains in trench fever are the most characteristic symptoms of the disease, although they are not always present. Similar pains are described in dengue fever, and in soldiers after long marches or after standing in mud or water for long periods.

There is no laboratory method which has a direct diagnostic value. Leukocytosis is usually found during the periods of pyrexia, but leukopenia is occasionally as marked as in typhoid fever or dengue.

The reason for reciting these confusing points is to show that a correct diagnosis is not readily made in a single examination or from the consideration of a few symptoms or signs. It is only by careful daily examinations and the recording of the findings, as well as by the elimination of other diseases, often by laboratory methods, that the largest proportion of correct diagnoses can be made.

The preceding remarks apply to the difficulties in making a diagnosis in every army under field conditions. Certain special difficulties existed in the American Expeditionary Forces. During the period when most of these cases occurred, we were passing through the influenza epidemic. It is usual for physicians to apply the name of the most prevalent acute disease to all pyrexias of undetermined origin that come under their observation. In this way they make the largest number of correct diagnoses, but at the same time many incorrect ones. Trench fever and influenza closely resemble one another at the onset. Many cases can never be differentiated. Most cases require prolonged observation and continuous temperature records. In the rapid movement of patients from one hospital

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to another, the temperature records frequently were not sent with them. The temperatures and pulse rates were often taken, but not recorded, because there was no convenient place to record them. It is self-evident that the field medical card should have a special blank for the recording of temperatures. Then the degree and the time of fever would be noted, and the medical officers in the hospitals in the rear would not be at a loss to know what had gone on in the forward areas. This knowledge is important for the proper care of both sick and wounded.

Influenza was constantly present in the American Expeditionary Forces, there being 190,689 cases reported from July, 1917, to May, 1919. In addition there were 21,939 cases of pyrexia of undetermined origin. It is easy to understand how a number of cases of trench fever might have been missed in these 212,628 cases.

### ETIOLOGY AND COURSE

Both the British and American Trench Fever Commissions have shown that the body louse is the chief agent in transmitting the virus from man to man. The British commission feels that the chief mode of inoculation is by the scratching of the feces of infected lice into the skin of louse infested men. The American commission showed that infection did occur following the bite of infected lice, even when there was no visible excoriation of the skin. The elucidation of the exact mode of inoculation is purely an academic problem, for it is certain that lice living under normal conditions can easily transmit the infection. The virus remains active for a long time in dry louse feces. It is also present in the urine of trench fever patients, its resistant qualities being shown by the fact that the concentration of such urine by drying does not kill it. It will pass through a Chamberland filter. Its thermal death point is above 60 C. All these characteristics show that it belongs to the group of resistant filterable viruses.

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The course of the disease in human beings is different from that ordinarily seen in most other acute infections. In many ways it resembles malaria. The number of relapses is variable. It tends to become subacute or chronic in many patients. In some there are long periods of latency, the patient appearing perfectly well, with later an acute exacerbation. There is no known specific cure. Recovery is evidently due to the development of some type of immunity. Lice may take up the virus from a patient weeks or months after the onset of the disease. Just as there is no known laboratory method of diagnosis, so there is no known method of determining when a patient may be no longer a source of infection.

## PROPHYLAXIS

From the foregoing observations, three factors must be considered in preventing the spread of the disease: (1) the louse as the transmitting agent; (2) the human foci of infection, and (3) the resistant nature of the virus.

The extent of lousiness of soldiers under field conditions is well known by many medical officers. Peacock reported that at least 95 per cent. of British soldiers living in the trenches were lousy. Many sanitary reports from our own army indicate a similar degree of infestation among American troops. Among the first sixty American volunteers who reported to the American Trench Fever Commission, 38.3 per cent. were infested, even though the entire detachment was supposed to have been recently deloused. It is remarkable how soon an entire division will become infested after an ordinary delousing. Constant, thorough inspection and easily accessible bathing facilities and delousing apparatus are necessary to keep all the troups of an army vermin free. It is needless to dwell on the general lack of these requisites during an active campaign.

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Our troops must have come into contact with many soldiers with trench fever. The disease was very prevalent in the British army. At least ten of our divisions spent a portion of their training period in the British area. Reports from American base hospitals attached to the British army show that a number of our soldiers were infected with the disease in this area, even though the proportion was not so high as among British troops. It is impossible to state accurately the degree of prevalence of the disease in the French army. We know, however, that it did exist. It was present to a considerable degree among the German soldiers, so that our advancing troops were exposed to infection in the captured trenches, and the troops of the army of occupation were exposed to a certain extent in German billets and barracks.

## INCIDENCE OF TRENCH FEVER IN THE AMERICAN EXPEDITIONARY FORCES

Table 1 shows that only a small number of cases of trench fever were reported in the American Expeditionary Forces. These figures are probably far from

TABLE 1.—TRENCH FEVER AND PYREXIA OF UNDETERMINED ORIGIN IN THE AMERICAN EXPEDITIONARY FORCES

| | Trench Fever | | Pyrexia of Undetermined Origin | | |
|---|---|---|---|---|---|
| | 1917 | 1918 | 1919 | 1918 | 1919 |
| January | .. | 3 | 188 | ..... | 2,476 |
| February | .. | 1 | 61 | ..... | 1,477 |
| March | .. | 3 | 51 | ..... | 703 |
| April | .. | 4 | 14 | ..... | 396 |
| May | .. | 0 | ... | ..... | ..... |
| June | .. | 19 | ... | 1,748 | ..... |
| July | .. | 33 | ... | 1,393 | ..... |
| August | 1 | 29 | ... | 1,552 | ..... |
| September | 2 | 67 | ... | 3,474 | ..... |
| October | 0 | 33 | ... | 3,759 | ..... |
| November | 3 | 15 | ... | 3,214 | ..... |
| December | 2 | 26 | ... | 1,747 | ..... |
| Subtotals | 8 | 233 | 314 | 16,887 | 5,052 |
| Totals | | | 555 | | 21,989 |

representing the actual number of cases. All of the reasons for making diagnosis difficult were constantly in operation. Many of the diagnoses were missed

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because of the rapid movements of the sick, but probably many more were missed because medical officers were not acquainted with the manifestations of the disease. This statement is borne out by the experience in the Third Army. Early in January, 1919, after the troops were in the areas they were to occupy permanently, and most of the sick were under continuous observation in the evacuation hospitals, a number of cases of typical trench fever were brought to light. An attempt was then made to acquaint all medical officers in the field and evacuation hospitals with the manifestations of the disease, the mode of transmission, and prevention. By means of informal reports, the medical officers of the various units from which cases came were urged to insist on better delousing measures.

Chart 3 shows the incidence and distribution of cases in the various units by weeks for the first three months of this year. All of the reports were made from the evacuation hospitals. In several of the cases reported between the 1st and the 11th of January, the onset occurred in December. In fact, many of the patients stated that their trouble began at the time of the march forward from the Argonne region into Germany. Taking the figures as they stand, however, we see that over three-fourths of the cases occurred in two divisions, the Forty-Second and the Second. The effect of delousing and removing patients from their division is well demonstrated, for after the third week in January the number of cases fell off rapidly in each division. The distribution of cases throughout the division, corps and army troops down to units as small as companies is shown in Tables 2, 3 and 4.

Cases were identified from 162 different companies. From each of 110 of these companies one case was diagnosed, from thirty-five companies two cases, from ten companies three cases, from five companies four cases, from one company five cases, and from one com-

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pany seven cases. Thus, while the infection was widespread throughout the army, it was not very extensive in any one unit. The figures suggest that foci of infection had been present among the troops only a short time. It is interesting to note, however, that the two

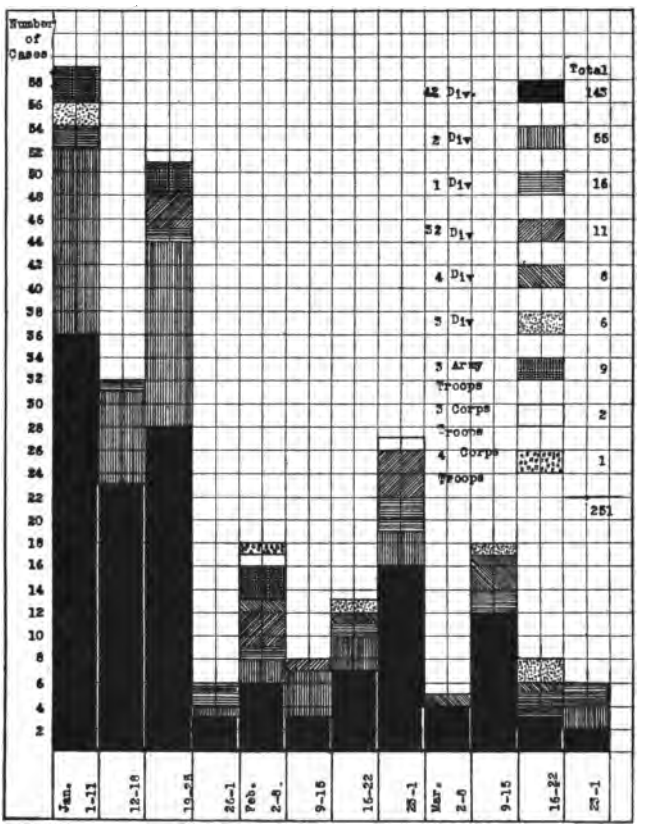


**Chart 3.—Incidence of trench fever in troops of Third Army, Jan. 1-March 1, 1919.**

divisions with the heaviest rate had been overseas since 1917. The lower rate in the other old division, the First, is in line with the general low sick rate in that division.

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TABLE 2.—DISTRIBUTION OF TRENCH FEVER CASES IN FORTY-SECOND DIVISION

| | 165th Infantry | | | | | | | | | | | | | | | | 149th Field Artillery | | | | | | | | | Military Police | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Company | A | B | C | D | E | F | G | H | 1 | K | L | M | M.G. | Hq. | Sup. | X | A | B | C | D | E | F | Hq. | Sup. | X | | | | | |
| Jan. 1-11 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | 1 | 1 | .. | .. | 1 | 1 | 2 | 1 | 3 | .. | .. | .. | .. | .. |
| 12-18 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. |
| 19-25 | .. | 1 | .. | 1 | .. | .. | .. | 1 | .. | 2 | 1 | 1 | .. | .. | .. | .. | 1 | .. | 1 | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. |
| 26- 1 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Feb. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. |
| Mar. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |

| | 166th Infantry | | | | | | | | | | | | | | | | 150th Field Artillery | | | | | | | | | 150th M. G. Bn. | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Company | A | B | C | D | E | F | G | H | 1 | K | L | M | M.G. | Hq. | Sup. | X | A | B | C | D | E | F | Hq. | Sup. | X | A | B | C | D | X |
| Jan. 1-11 | .. | .. | .. | 2 | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 |
| 12-18 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. |
| 19-25 | .. | .. | .. | 2 | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 26- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 |
| Feb. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 4 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | 1 |
| 23- 1 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 2 | .. | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. |
| Mar. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |

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| Company | 167th Infantry | | | | | | | | | | | | | | | | 151st Field Artillery | | | | | | | | | 151st M. G. Bn. | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | A | B | C | D | E | F | G | H | I | K | L | M | M.G. | Hq. | Sup. | X | A | B | C | D | E | F | Hq. | Sup. | X | A | B | C | D | X |
| Jan. 1-11 | . | .. | 2 | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | 2 | .. | .. | 1 | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. |
| 12-18 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | 4 | .. | .. | .. | 1 | .. |
| 19-25 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | .. |
| 26- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Feb. 2- 8 | .. | . | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. |
| Mar. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | 1 | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |

| Company | 168th Infantry | | | | | | | | | | | | | | | | 117th Engineers | | | | | | | | | 117th Fd. Sig. Bn. | 117th Amm. Tr. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | A | B | C | D | E | F | G | H | I | K | L | M | M.G. | Hq. | Sup. | X | A | B | C | D | E | F | Hq. | Sup. | X | | | | |
| Jan. 1-11 | . | 2 | .. | 3 | .. | 1 | 1 | 1 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. |
| 12-18 | . | .. | 1 | .. | 1 | .. | .. | .. | .. | 2 | .. | 3 | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .1 | .. | 1 | .. | .. | .. |
| 19-25 | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | 1 | 1 | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. |
| 26- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Feb. 2- 8 | .. | 1 | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | . | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. |
| Mar. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |

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TABLE 3.—DISTRIBUTION OF TRENCH FEVER CASES IN SECOND DIVISION

| | 9th Infantry | | | | | | | | 12th Field Art. | 23d Infantry | | | | | | | | | | 15th Field Art. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Company | A | B | C | D | H | L | M.G. | Hq. | F | C | D | E | F | G | I | L | M.G. | Hq. | Sup. | F | Hq. | Sup. |
| Jan. 1-11 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | 1 | .. | .. | 1 | 1 | 2 | .. | .. | 1 | .. |
| 12-18 | .. | 1 | .. | .. | .. | .. | 1 | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 19-25 | 1 | .. | 1 | 1 | 1 | 1 | 1 | .. | .. | .. | .. | .. | 2 | .. | .. | 1 | .. | .. | .. | .. | .. | .. |
| 26- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. |
| Feb. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | 1 |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | . | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. |
| Mar. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. |

| | 5th Marines | | 2d Engineers | 1st Field Sig. Bn. | 17th Field Art. | | | | 6th Marines | | | | | 2d Amm. Tr. | | 2d San. Tr. | | 6th M. G. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Company | 17 | 49 | | | C | F | Hq. | X | 15 | 75 | 78 | 95 | X | B | D | 16th Amb. | 22d Fd. Hosp. | X |
| Jan. 1-11 | 1 | .. | .. | 1 | .. | 1 | 1 | 1 | .. | 1 | .. | .. | .. | .. | 1 | 1 | 1 | .. |
| 12-18 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | 1 | .. | 1 | .. | .. | .. | .. | 1 |
| 19-25 | .. | .. | .. | 2 | .. | 1 | 1 | .. | 1 | .. | .. | 1 | .. | 1 | .. | .. | .. | .. |
| 26- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Feb. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | 2 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | 1 | .. | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |

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TABLE 4.—DISTRIBUTION OF TRENCH FEVER CASES IN FIRST, THIRD, FOURTH AND THIRTY-SECOND DIVISIONS, THIRD CORPS, FOURTH CORPS, AND THIRD ARMY TROOPS

| | 1st Division | | | | | | | | | | | | | | | 32d Division | | | | | | | | | 3d Corps Troops | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 16th Inf. | 28th Inf. | | | 18th Inf. | | | | | 6th F. A. | | 7th F. A. | | 1st Amm. | P. E. | 125th Inf. | | | 126th Inf. | 127th Inf. | | | | 128th Inf. | 323d F. A. | 308th Eng. | 146th F. A. |
| Company | K | B | F | M.G. | C | E | F | H | L | A | Hq. | D | F | Tr. | S. | C | F | X | M | F | H | K | Hq. | C | F | C | B |
| Jan. 1-11 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 12-18 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 19-25 | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | 1 | 1 | .. | .. | .. | 1 |
| 26- 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Feb. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | 1 | 1 | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | 2 | .. | 1 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. |
| Mar. 2- 8 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15 | .. | .. | .. | .. | .. | .. | .. | .. | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22 | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1 | .. | .. | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |

| | 3d Division | | | | 4th Division | | | | | | 4th Corps Troops | 3d Army | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 4th Inf. | 7th Inf. | 6th Eng. | | 76th F. A. | 39th Inf. | 47th Inf. | | | 58th Inf. | | 11th M.G. Bn. | 51st Pion. | 1st Pion. | | 3d Army Troop | Hq. Bakers | Evac. Hosp. 4 | S. C. 1 | A. T. C. 4 | 149th Inf. (?) | 327th F. A. (?) |
| Company..... | D | Hq. | B | E | D | X | D | F | I | F | I | | | A | K | | | | F | | | |
| Jan. 1-11..... | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | 1 | .. |
| 12-18..... | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 19-25..... | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | 1 | .. | .. | .. | 1 |
| 26- 1..... | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Feb. 2- 8..... | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | 1 | 1 | .. | .. | .. | .. | .. |
| 9-15..... | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22..... | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1..... | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Mar. 2- 8..... | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 9-15..... | .. | 1 | .. | .. | .. | .. | 2 | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 16-22..... | .. | 1 | 1 | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 23- 1..... | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |

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By the first of February, delousing facilities were fairly adequate. This month the rate dropped to less than half that of January, and in March it was about one fourth the January rate. The continuation of the disease in February and March was probably due in part to infection incurred in January, and in part to infection contracted elsewhere. At this time, large numbers of men were going on leave to various parts of France where troops from other units of the American Expeditionary Forces were also on leave. Leave, with the diminished medical supervision and the necessary crowding on trains and in billets, was a fruitful source of infection of all kinds.

A comparison of the number of reported cases (300) in the entire American Expeditionary Forces in the first three months of 1919 with the number from the Third Army for the same period reveals the fact that five sixths of the reported cases were from the Third Army. In spite of the heavy incidence in two of the divisions, it is highly improbable that one seventh of the entire American Expeditionary Forces would furnish five sixths of the cases, especially when several of the divisions then in France had been more exposed to trench fever. We are, therefore, forced to conclude that a considerable number of cases existed throughout the American Expeditionary Forces that were never recognized.

### POSSIBILITY OF SPREAD

The return of so many soldiers to civil life will doubtless result in distributing foci of infection throughout the country. Every possible precaution is being taken to see that the troops are completely deloused before embarkation and demobilization. Nevertheless, the possibility of long latent periods and late relapses, as well as the power of body lice to take up the virus during the late periods of the disease, must be borne in mind. Lice are not confined to

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armies. In our cities there is constant danger of transmission of any louse-borne disease if human foci of infection exist.

The fact that trench fever is not fatal has naturally caused it to be disregarded when there were so many more urgent conditions demanding the attention of the medical officers. As an economic problem, it must be recalled that, on the average, a trench fever patient is incapacitated for six weeks. Often the infection persists for months. The effort syndrome is a not infrequent sequela; indeed, this symptom may be an indication of the persistence of the infection. It is, therefore, important to keep in mind, as after-war problems, both the possibility of the spread of the infection and its effect on discharged soldiers.

## ABSTRACT OF DISCUSSION

DR. WILLIAM S. THAYER, Baltimore: The only way to learn the practice of medicine is by practice and experience, and there is no greater demonstration of that than what has been shown in the paper by Dr. Swift. Dr. Swift went up into the active area and found trench fever; the rest of us did not find it.

DR. ERNEST ZUEBLIN, Cincinnati: I think any new disease of which we have heard so much is liable to arouse our curiosity, and I think we are really fortunate to have Dr. Swift tell us about his experience. With the discussion of a new disease such as trench fever, many questions arise which I would not like to touch on at the present moment, but first I would like to know whether in addition to the cardiac manifestations any other syndromes have been encountered; second, whether the means of controlling the lice have been sufficient to prevent any further propagation of the disease; and third, how long a time is required, that is, for how long a time the patient is still to be considered as infective and a danger to the community, and what are the means available to determine the extent and duration of a cure from the disease.

DR. HOMER F. SWIFT, New York: I forgot to mention that the disease is not fatal. We do not know of any patient who died as a result of trench fever. I think in the British army a large proportion, 50 per cent., of the cases of D. A. H., had trench fever. Another late symptom is the persistence of leg pains and lumbar pains. Many of these patients come in with the diagnosis of chronic rheumatism or chronic lum-

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bago, and when you go back and reconstruct their histories, you will find that they began with definite relapses every few days. Unfortunately, we have no way of telling how long a patient will remain a source of infection. We have no laboratory method of diagnosing the disease, and we have no way of telling how long that patient will infect a louse before a louse will infect a human being. I think as long as a patient is showing active symptoms of the disease he should be a subject for isolation, not strict isolation as in measles, but isolation from infestation.

The second point as to the amount of heat necessary to kill the virus, you will see that from 56 to 60 C. kills the lice and also kills the eggs, so that for ordinary purposes heating clothes at 60 C. is sufficient to delouse. That is not sufficient to kill the virus in the feces of the lice or the virus in the clothes that may have come from urine or saliva, so that it takes a higher degree of heat to disinfect clothes which have come into contact with trench fever patients, as well as disinfect them.

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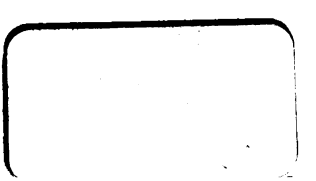

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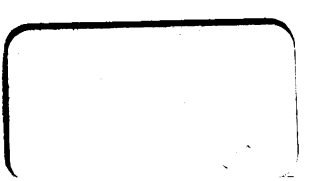

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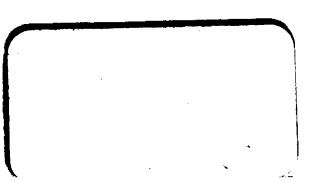

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